University of Alberta

Effect of Elk Velvet Antler Supplementation During Concurrent Training in Rowers

by

Kirsten MacFadyen

A thesis submitted to the Faculty of Graduate Studies and Research in partial fulfilment of the requirements for the degree of Master of Science

Department of Physical Education and Recreation

Edmonton, Alberta

Spring 2003

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ABSTRACT

The purpose of this study was to examine the effects of elk velvet antler supplementation (EVA) on adaptations to concurrent resistance and endurance (3x/wk each) training. Forty-five volunteers performed physiological testing before and after 5 and 10 weeks of training. Measurements included VO_2 max, 2000 m rowing time, leg and bench press strength. Blood serum hormone levels were measured at rest and 5 and 60 minutes after the 2000 m test. Subjects ingested either EVA (560 mg/day) or placebo capsules. Significant ($p<.05$) increases occurred in both groups for VO_2max and strength. 2000 m rowing time significantly decreased to the same extent in both groups. There was a significant increase in testosterone, growth hormone and cortisol after the 2000 m test but no changes were observed in resting hormone levels or after training for either group. It appears that 10 weeks of EVA does not significantly improve various physiological or performance parameters greater than training alone.

ACKNOWLEDGEMENTS

There are two people I could not have completed this thesis (and degree) without: Dan Syrotuik and Gord Bell. I am forever indebted to you both for your support and encouragement along the way. You stood behind me and had belief in me, even when I didn't always believe in myself.

Thank you Tuck, for being such a great advisor and for always making sure that I made time for the important things in life (golf). You taught me that having a career is different from having a life, and for that I will always be grateful. A person could not ask for a better supervisor. One with a better golf handicap, perhaps, but not a better advisor! Remember, as long as you finish with more golf balls than you started with, it's been a good game.

Gord, what can I say? Without you, I wouldn't have had a study to complete. Thank you for allowing me to use your annual rowing study for a thesis. You came to my rescue exactly when I needed it. One evening in desperation I threw a coin in the treasure chest of the lagoon at West Edmonton Mall and made a wish that I would get a new thesis topic. The very next day you offered me your rowing study. You truly did answer my prayers! I thank you for your all of your help along the way. I wouldn't be where I am today if not for you. Your incredible knowledge and experience, combined with your enthusiasm and dedication to the field, both as a researcher and practitioner, are inspiring. Never lose that, you make physical education/kinesiology a great field to be in.

Lastly, I could not have gotten through this without the support of my friends. Alex Game, you've helped me out in more ways that I could possibly list. Your support, assistance, encouragement, and employment have kept me going throughout the years,

academically, professionally and personally. Without you, I may never have known the joys of an afternoon drinking at RATT, the cinnamon buns in Education, and the best golf courses in Edmonton. You have also helped me along the way by providing me with a wide variety of testing and professional development opportunities. I count you among my greatest friends and know that I always have a staunch supporter in you. Likewise.

Tony Webster and Kelly Mackenzie, two of my other greatest friends, I could not have gotten to where I am today without you. A person meets a lot of great people in Edmonton! Tony, you taught me a lot of life lessons along the way, but most importantly you taught me how to golf – a sport in which I will need lessons all my life! I'm forever grateful for your friendship. Kelly, you've always been there when I needed you. You provide me with a shoulder to lean on and a bed to sleep in when I need it. I only hope that someday I will be able to help you out as much as you've helped me. You're fantastic Kel, don't ever change.

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CHAPTER 1

INTRODUCTION

1.1 Introduction

Rowing is a sport that demands high levels of both strength and endurance to be successful (Secher, 1993). The physiological basis to perform the sport is primarily developed through strength and aerobic endurance training. However, some researchers have suggested that an "interference effect" in strength development may occur as a result of simultaneous strength and endurance training (Hickson, 1980; Bell et al, 2000), such that the effectiveness of the strength training is compromised. The mechanisms behind this interference effect are becoming clearer. Leveritt and colleagues (1999) propose an acute hypothesis, contending that residual fatigue from the endurance training component compromises the ability to develop force or tension during the strength element of concurrent training, thereby preventing optimal strength development and adaptation from occurring.

Hickson (1980) speculates that the impairment of strength development, but not to endurance, during concurrent training may be directly related to the muscles inability to adapt to both forms of exercise, as the adaptations to strength and endurance training are divergent in nature. Research by Bell et al. (2000) supports the contention that combined strength and endurance training can suppress some of the adaptations to strength training and augment some aspects of capillarization in skeletal muscle, which is a common adaptation to endurance training. Other research by Bell et al. (1991) suggests that the interference of strength development is due to a lack of muscle fibre hypertrophy.

A further explanation for the interference effect on strength development during concurrent strength and endurance training suggests that concurrent training may hinder organization of efficient motor unit recruitment patterns necessary for forceful muscular contractions (Chromiak & Mulvaney, 1990; Dudley & Djamil, 1985). A final explanation may be related to an increase in cortisol as a result of training (Bell et al., 1997; Kraemer et al., 1995), which has been demonstrated to inhibit testosterone release (Cumming, 1987). Anabolic hormones such as testosterone and growth hormone are critical in maintenance and building of skeletal muscle mass as a result of strength training (Kraemer et al., 1995; Bell et al., 2000). Thus, if anabolic hormones are inhibited and/or catabolic hormones are elevated strength development may be compromised. Therefore, further strategies must be found to avoid a potential interference effect in strength development for athletes performing concurrent strength and endurance training and enable them to optimize the training benefits.

Athletes and commercial supplement companies have made many claims about the effects of antler velvet that may work to counteract the interference of strength development with concurrent training. Velvet antler has been said to aid performance, boost stamina, strength and efficiency, and work to increase muscle mass. Velvet antler has been used as a traditional medicine in the Orient for thousands of years, for ailments such as arthritis and infertility, however the mechanisms by which it may aid athletic performance are not known. Velvet antler from Wapiti (*Cervus elaphus*) have been shown to contain a variety amino acids, lipids, ash, collagen, calcium, phosphorus, magnesium, sialic acid, uronic acid and sulfated glycosaminoglycans (GAGs) (Sunwoo et al., 1995). It is also thought that elk velvet antler (EVA) may contain hormones,

prostoglandins and growth factors, possibly providing steroid hormone precursors that contribute to an anabolic hormone environment.

World-class athlete New Zealand rower Rob Waddell attributes part of his successful performance to velvet supplementation, stating,

> "My ability to handle a higher load of training, and the level of my strength and physiology have been distinctly improved for the base period of training in which I was trialing deer velvet (The Deer Farmer, New Zealand; www.velvet.org.nz/news.cfm; http://www.sexualboost.com/index.html)."

Hamish Carter, the 1998 World Triathlon Champion, says,

> "Since taking deer velvet I found that my recovery from training, competition and even travel has been greatly enhanced. I feel much more on top of my training schedule and this gives me confidence when I race…I have been taking 1200 mg of velvet daily for 6 months and believe it has helped me in giving the endurance to be competitive day in and day out on the World Cup Triathlon Circuit (www.impaxsupply.com/sports.htm; www.velvet.org.nz/raves.cfm)."

Yet, despite the accolades by such accomplished athletes, little peer-reviewed literature exists to substantiate these claims.

1.2 Purpose

Therefore, the purpose of this study is to examine the effects of EVA supplementation on measures of rowing performance and other physiological parameters after 5 and 10 weeks of combined resistance and endurance training. Measures of VO_2 max, 2000 m rowing performance time, estimated one repetition maximum bench and leg press strength and serum hormone levels (free testosterone, total testosterone, human growth hormone and cortisol) will be assessed on volunteers from the Edmonton (Alberta, Canada) rowing community prior to commencing training, after 5 weeks of training and following completion of the 10 week training program.

1.3 Hypothesis

It is hypothesized that subjects who supplement with EVA while training will have enhanced performance over subjects training with a similar program, but ingesting a placebo. As a result, subjects who supplement with EVA will likely have an enhanced anabolic profile when compared to the placebo group, enabling them to maintain strength development while performing concurrent strength and endurance training.

1.4 Significance of the Study

As a result of an enhanced anabolic environment, supplementing with EVA could help athletes gain the extra boost they need during intense training to overcome any possible interference effect of strength development found during concurrent strength and endurance training. The results of this study may broaden the application of velvet antler as an ergogenic aid to enhance physical fitness training to a wide variety of athletes and recreational fitness enthusiasts alike. Thus, it is important to examine the potential of velvet antler supplementation to create an anabolic environment and enhance human performance.

The results of this study will provide more information on the relationship between EVA supplementation in an athletic setting and on changes in standard training hormone measures such as testosterone, cortisol and human growth hormone. By examining these relationships, this study will provide insight on the efficacy of using EVA as an ergogenic aid and its resulting influence on performance. Further, if EVA is found to promote an anabolic environment it may have potential to be useful in clinical rehabilitation situations where muscle wasting has occurred.

1.5 Delimitations

- The study was delimited to volunteers with rowing and strength training experience. A total of 45 volunteers (25 males, 20 females) with varied amounts of rowing and strength training experience participated.
- Subjects were required to limit their activity to the training program provided. Extra training and sport involvement was subject to approval by the principal investigator, Gordon Bell, Ph.D. All subjects performed the same training program, consisting of three days of endurance training and three days of strength training per week, for 10 weeks.
- The study was delimited to common measures of athletic performance, including VO_2 max, predicted one repetition maximum leg and bench press strength, and 2000 m rowing time. VO_2 max measurements were delimited to those that the Medgraphics CPX/D metabolic cart (Medical Graphics, St. Paul, MN) is capable of measuring. VO_2 max and 2000 m rowing tests were performed by all subjects on Concept II rowing machines. Leg press was measured on a Universal incline (45°) leg press machine, with a sled weight of 84.1 kg (185 pounds). Bench press was performed on a flat bench using an Olympic bar and standard weights.
- Testing was performed prior to, midway, and following a 10-week training program. All subjects performed a VO_2 max test, 2000 m rowing test and multiple repetition maximum (MRM) strength test. Each test was separated by a minimum of 24 hours rest. The study was delimited to subjects completing all tests during each of the three testing sessions.

- The study was delimited to subjects not consuming any other supplements during the course of the study.
- Blood measures during the study were delimited to control for the natural circadian rhythm of the hormones. All resting blood measures were taken at the same time of day to control for natural hormone cycles.
- The study was a randomized, double blind, placebo-controlled, pre-, mid- and post-test design. Subjects either consumed EVA capsules or placebo capsules designed to look like the EVA capsules, and contained in identical bottles. All EVA was supplied by Royal Elk Products (Sangudo, Alberta) and was produced in the same lot number (Lot #120).
- The independent variable in the study is the EVA supplementation. The EVA supplementation was controlled in both quantity and frequency. The dependent variables are the performance measures: VO_2 max, 2000 m rowing time, and MRM leg and bench press scores. Other dependent variables are measures of free testosterone, total testosterone, cortisol and growth hormone.

1.6 Limitations

- Motivation of the subjects could be a limiting factor in the study. While investigators gave encouragement to all subjects, individual motivation of the subject could limit the effort to which the testing and workouts were performed.
- Volunteers were invited to participate in the study, given that they had prior rowing and strength training experience, in order to allow for an adequate sample size. This precludes random selection of subjects and may have introduced a bias

in the subject selection as only those who were motivated to perform the intense testing and training schedule would have volunteered for the study.

- Experimenter and equipment error may have limited the study.
- The study was limited to a 10-week training program due to the dates of the Winter Term at the University of Alberta and the time required to complete all of the testing. Thus, it is possible that not all subjects were able to achieve maximum results in the 10 weeks allowed for training.
- The study was limited in the fact that dietary records were not analysed for subjects participating in the study. It is possible that any effects observed in the study, or lack thereof, could be as a result of changes in diet and not a factor of the training program.
- The study was limited in the dosage of EVA given to participants. As no dose-response study has been conducted using EVA, we chose to follow the manufacturer's recommendations for dosage. It is possible that the manufacturer's recommendation for dosage of EVA is not enough to elicit a significant training response.

1.7 Definitions

$V0_2$ max The rate of oxygen uptake during maximal aerobic exercise, reflects 1) the capacity of the heart, lungs and blood to transport oxygen to the working muscles, and 2) the utilization of oxygen by the muscles during exercise. Maximal and submaximal VO_2 is expressed in absolute or relative terms. Absolute VO_2 is measured in litres per minute ($L \cdot min^{-1}$) or millilitres per minute ($ml \cdot min^{-1}$) and provides a measure of energy cost for non-weight-

bearing activities such as leg or arm cycle ergometry. Absolute VO_2 is directly related to body size; thus men typically have a larger absolute VO_2 max than women. To compare individuals who differ in body size, VO_2 is expressed relative to body weight ($ml \cdot kg^{-1} \cdot min^{-1}$). Relative VO_2 is used to estimate the energy cost of weight-bearing activities such as walking, running, stepping, dancing and stair climbing. Directly measured maximum oxygen uptake (VO_2 max) or peak VO_2 is considered to be the most valid measure of functional capacity of the respiratory system (Heyward, 1998).

Strength The ability of a muscle group to develop maximal contractile force against a resistance in a single contraction (Heyward, 1998).

Multiple Repetition Maximum (mRM) The maximum weight that can be lifted for a determined number of complete repetitions of the movement, i.e. 8 RM (Heyward, 1998).

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CHAPTER 2

REVIEW OF LITERATURE

2.1 Introduction

To be successful in many sports and activities, including rowing and military training, individuals demand high levels of both strength and endurance. Adaptations from strength and endurance training are often opposite in nature, such that the development in one may be compromised by simultaneously training both, resulting in an "interference effect." This literature review will discuss the physiology of rowing and the interference effect. Further, this review will examine the effects of acute and chronic exercise on testosterone, growth hormone and cortisol, hormones that are identified as being important in characterizing the training environment as anabolic or catabolic. In addition, research problems and concerns when investigating hormones will be discussed. Finally, elk velvet antler (EVA) will be introduced, its properties and dosages described, and the relationship between velvet antler supplementation and exercise will be examined.

2.2 The Interference Effect

Rowing is a sport that demands high levels of both strength and endurance to be successful (Secher, 1993). The physiological basis to perform the sport is primarily developed through strength and aerobic endurance training. However, some researchers have suggested that an "interference effect" in strength development, but not to endurance, may occur as a result of simultaneous strength and endurance training (Bell et al., 1997; Bell et al, 2000; Dudley & Djamil, 1985; Hennessy & Watson, 1994; Hickson, 1980; Kraemer et al., 1995), such that the effectiveness of the strength training is

compromised. However, support for this belief has not been universal (McCarthy et al., 1995; McCarthy et al., 2002; Nelson et al., 1990; Sale et al., 1990). As Sale et al. (1990) note, whether an interaction occurs and the form it takes likely depend on several factors, including: The intensity, volume and frequency of the strength and endurance training; the modes of training; the fitness of the subjects; and, the method in which the two forms of training are integrated. The length of time of the training study may also result in different interaction results (Nelson et al., 1990). It has been argued that the interference effect observed by some researchers may be due to possible overtraining of the subjects involved in the study (Dudley & Djamil, 1985; Dudley & Fleck, 1987; Hickson, 1980). Furthermore, the results and conclusions drawn by researchers will be affected by the selected criterion measures of strength and endurance development, and must be taken into consideration when interpreting the results of research studies (McCarthy et al., 1995; Sale et al., 1990).

The mechanisms behind the interference effect of simultaneous strength and endurance training are unclear, although some researchers (Chromiak & Mulvaney, 1990; Dudley & Fleck, 1987; Hickson, 1980) postulate that the impairment of strength development, but not to endurance, during concurrent training may be directly related to the muscles inability to adapt to both forms of exercise, as the adaptations to strength and endurance training are divergent in nature. Research by Bell et al. (2000) supports the contention that combined strength and endurance training can suppress some of the adaptations to strength training and augment some aspects of capillarization in skeletal muscle, which is an adaptation to endurance training.

Hickson (1980) speculates that the marked impairment of strength development during concurrent training may be a result of the residual fatigue due to the training volume involved with simultaneous strength and endurance training. Evidence in the results of Hickson's study contradict this speculation, however, as both the endurance groups and the concurrent training groups developed endurance gains at the same rate throughout the study, even while the strength gains in the concurrent training group were decreasing toward the end of the training period. Thus, it appears that the effects on strength development are selective.

Leveritt and colleagues (1999) propose an acute hypothesis for the interference effect, contending that residual fatigue from the endurance training component compromises the ability to develop force or tension during the strength element of concurrent training, thereby preventing optimal strength development and adaptation from occurring.

Other research (Bell et al., 1991; Kraemer et al., 1995) infers a more chronic hypothesis in that the interference of strength development is due to a lack of muscle fibre hypertrophy. This hypothesis is supported by subsequent research by Bell et al. (2000), in which it was found that type II fibres gained size following both strength training and concurrent strength and endurance training, while type I fibres did not change in cross-section area following concurrent training. The researchers postulated that this result may have been due, in part, to the oxidative stress imposed on the muscle and the need to optimize the kinetics of oxygen transfer as a result of the addition of endurance training to strength training.

An alternate explanation for the interference effect on strength development during concurrent strength and endurance training suggests that concurrent training may hinder organization of efficient motor unit recruitment patterns necessary for forceful muscular contractions (Chromiak & Mulvaney, 1990; Dudley & Djamil, 1985). Further, it has been suggested that one of the factors in the compromised strength gains may be due to shifts in skeletal muscle myosin isozymes. Fibre type transformation from fast to slow myosin isozymes may be possible with prolonged and intense endurance training hindering maximal power output ability (Chromiak & Mulvaney, 1990). A training-induced transformation of fibre types profiles from slow to fast is much more difficult to achieve, as slow twitch fibres are continuously stimulated by the typical low frequency pattern of the slow motor neuron between training sessions (Chromiak & Mulvaney, 1990). This continuous influence is much stronger than any stimulation during training, thus changes induced by strength training are restricted to adaptations in metabolic function and fibre size (Chromiak & Mulvaney, 1990).

A final explanation for the interference effect may be related to an increase in cortisol as a result of training (Bell et al., 1997; Bell et al., 2000; Kraemer et al., 1995, Leveritt et al., 1999), which has been demonstrated to inhibit testosterone release (Cumming, 1987). Anabolic hormones such as testosterone and growth hormone are critical in maintaining and building skeletal muscle mass as a result of strength training (Bell et al., 2000; Kraemer et al., 1995). Thus, if anabolic hormones are inhibited, strength development may be compromised.

Bell et al. (2000) examined the hypothesis that if compromised strength gains occurred as a result of strength training being combined with endurance training, there

may be a higher catabolic state as evidenced by elevated cortisol concentrations and a lack of change in muscle fibre size. The researchers randomly assigned 45 volunteers by gender into one of four groups: strength training only (n=11), endurance training only (n=11), combined training (n=13), and a control group (n=10). The results of the study provided evidence of a reduced change in knee extension strength in the combined training group when compared to the strength only group; a lack of skeletal muscle hypertrophy, particularly in type I fibres, compared to the strength only group; and, an increased level of urinary cortisol as a result of concurrent training. Thus, this study found that concurrent strength and endurance training resulted in adaptations that differed from either strength or endurance training alone. Bell and colleagues (2000) further noted that urinary cortisol concentrations at rest were significantly elevated in women in the combined training group. In addition, men in the endurance-training group demonstrated a decrease in urinary cortisol concentrations, while men in the combined training group did not show a difference. These results, coupled with no changes in serum testosterone or growth hormone, suggest that concurrent strength and endurance training can lead to an elevated catabolic state in both men and women, which may compromise the effects of strength development during concurrent training.

While it is unclear the reasons why an interference effect in strength development may occur with concurrent strength and endurance training, many researchers (Bell et al., 1997; Bell et al., 2000; Dudley & Djamil, 1985; Hennessy & Watson, 1994; Hickson, 1980; Kraemer et al., 1995) agree that strength development appears to be compromised. The reason for this compromised strength development may simply be due to residual fatigue that occurs with both forms of training. Craig et al (1991) suggest that residual

fatigue from the endurance training component may compromise the ability of the muscle to generate tension during the strength element of concurrent training. Since the ability of a trainee to generate maximal muscle tension during a strength training session is critical in producing optimal strength development, the inability to produce such tension may impair strength gains. Nonetheless, further strategies must be found to minimize the potential interference effect in strength development for athletes performing concurrent strength and endurance training to enable them to optimize the training benefits.

2.3 Measures of Stress

2.3.1 Testosterone

Testosterone is an androgen, or male sex hormone. Androgens are gonadocorticoids, which are sex hormones produced in the gonads and in the zona reticularis of the adrenal cortex. The amount of sex hormones produced in the adrenal cortex is insignificant compared to the amounts produced by the gonads (Marieb, 1995). In males, the primary production of androgens takes place in the Leydig cells of the testes (Marieb, 1995; Hackney, 1996). The primary testicular androgen is testosterone, which is produced from cholesterol in the smooth endoplasmic reticulum (see Cumming et al., 1989, Fig. 1 for the complete pathway). Andrenogenesis is primarily regulated by luteinising hormone (LH) (Cumming, 1989) in the hypothalamic-pituitary-gonadal (HPG) axis for regulation of the male reproductive system. Periodically, the hypothalamus releases pulses of gonadotrophin-releasing hormone (GnRH) into the hypophyseal circulation, which supplies the hypothalamus and the anterior pituitary. The GnRH stimulates the anterior pituitary to produce and release LH and follicle-stimulating hormone (FSH) (Hackney, 1996). FSH, along with a small proportion of testosterone, is

responsible for spermatogenesis, the primary function of the male reproductive system (Cumming et al., 1989), an issue that is beyond the scope of this literature review.

GnRH controls LH synthesis and secretion. The pulsatile release of GnRH results in LH also being released in a pulsatile fashion into the system circulation (Hackney, 1996; Hoffman, 1992). In normal healthy males, approximately 2 to 4 LH pulses are observed during a 6 to 8 hour period (Hackney, 1996). At the testicles, LH interacts with its target tissue, receptors located on the cell membrane of the Leydig cells (Hackney, 1996). Once a hormone-receptor complex is formed in the Leydig cells, there is a mobilization of steroid precursors, particularly the activation of pregnenolone synthesis from cholesterol. Pregnenolone serves as the parent compound from which testosterone is derived. The anterior pituitary hormone prolactin, in normal concentrations, also acts to enhance the action of LH at the Leydig cells (Hackney, 1996). The circulating concentration of the various HPG axis hormones is a function of the amount of hormone produced and secreted into the blood pool and the amount leaving, usually through metabolic clearance (Hackney, 1996).

In circulation, testosterone is bound to a specific, high-affinity plasma protein, sex-hormone-binding globulin (SHBG), and has a low affinity to albumin, which has a large binding capacity (Cumming, 1987; Cumming et al., 1989). A small amount of testosterone circulates as the free steroid. The binding of testosterone to SHBG decreases its clearance, thus it seems likely that the biologically active portions of testosterone are the small, unbound portion and the portion bound to albumin (Cumming, 1987; Cumming et al, 1989). The circulating concentration of testosterone peaks in the morning between 6 a.m. and 8 a.m., then declines by approximately 35 % before beginning to rise again at

around midnight (Hoffman, 1992). Testicular testosterone production in the average male is approximately 7000 *ug* per day (Hackney, 1989), with normal resting testosterone values for mature males ranging from 14.0 to 28.0 $nmol \cdot L^{-1}$ (Hoffman, 1992). Other reported normal values range from 1.88 to 8.96 $ng \cdot ml^{-1}$ with a mean of 5.44 $ng \cdot ml^{-1}$ for males and from undetectable levels to 0.62 $ng \cdot ml^{-1}$ with a mean of 0.28 $ng \cdot ml^{-1}$ for females (DiaSorin Inc.).

Testosterone has many effects on reproductive organs and the promotion of secondary sex characteristics. Testosterone is also responsible for neural and metabolic effects. With regards to metabolic effects, testosterone has overall anabolic effects and promotes haematopoiesis, or blood cell formation, as well as enhancing the basal metabolic rate and stimulating protein synthesis (Marieb, 1995).

Exercise associated responses of the HPG axis are dependent upon the intensity and duration of the activity, as well as the fitness of the individual (Cumming et al, 1989). In general, relatively short bouts of high intensity exercise produce an increase in circulating testosterone, while more prolonged exercise tends to reduce testosterone levels. With prolonged exercise, testosterone may be inhibited for considerable time beyond the completion of the exercise (Cumming et al., 1989).

Kraemer et al. (1998) demonstrated an increase in testosterone levels following resistance exercise training. An increase in concentrations of testosterone was observed in both men and women after 6 and 8 weeks of training. This suggests that basal testosterone levels are increased in both men and women even after short-term resistance training, although men have significantly higher testosterone concentrations than women at all time points (Kraemer et al., 1998). The researchers suggest that these increases are

likely to be coupled to the tissue growth/remodelling process that is known to occur during a resistance program. Kraemer and colleagues (1998) further observed in their study that men were able to develop an acute exercise-induced increase in testosterone concentration with resistance exercise. These findings are supported by the work of Hakkinen et al. (1998), who observed that both total and free testosterone increased in men following heavy resistance isometric exercise, but that the response of testosterone to heavy resistance isometric exercise may be lowered with increasing age. To what extent the acute changes in the hormone environment created repeatedly during resistance training are related to long-term anabolic adaptations contributing to training-induced muscular hypertrophy and strength development is not well known (Hakkinen et al., 1998).

Researchers have shown significantly increased testosterone concentrations after short-term submaximal exercise (Hackney, 1996). Furthermore, it has been demonstrated that during a graded exercise bout to exhaustion, testosterone concentrations increase somewhat proportionally to the intensity of the exercise (Cumming et al., 1986; Galbo et al, 1977). Galbo and colleagues (1977) also found that the peak testosterone responses coincided with the maximal exercise intensity reached, then declined during the recovery period following exercise.

Submaximal exercise testosterone responses are dependent on the duration and intensity of the exercise (Hackney, 1989) as well as the fitness status of the individual (Vogel et al., 1985). During prolonged submaximal exercise to exhaustion, testosterone rises initially then declines as the activity continues (Galbo et al., 1977; Hackney, 1996). The reduction in the testosterone is typically in the magnitude of 25 to 50 % for activity

of 2 hours or longer duration (Hackney, 1996). Concentrations have been found to return to normal range within 24 to 72 hours after the activity (Hackney, 1996). Jensen and colleagues (1991) conducted a study comparing changes in testosterone concentrations after strength and endurance training in well-trained men, and observed that the changes in mean testosterone values followed the same time course after single sessions of strength and endurance training of the same duration and perceived exertion.

Vogel et al. (1985) found that both free and total serum testosterone levels increased significantly in untrained males during moderate exercise. The researchers exercised males at 50 % of their VO_2 max on a cycle ergometer for 45 minutes and found that while each subject reached peak testosterone levels at different times, serum testosterone levels were significantly increased over resting values after 15 minutes of exercise.

There is some debate over the primary physiological mechanism that accounts for the changes in testosterone in response to maximal and submaximal exercise (Hackney, 1996). Some of the proposed explanations have been alterations in hormonal synthesis at the endocrine gland, protein binding, metabolic clearance rate, haemoconcentration, sympathetic stimulation, GnRH pulse release and testicular blood flow (Hackney, 1996). It is not conclusive as to which of these factors is the exact mechanism of the testosterone change to acute bouts of maximal and submaximal exercise, but is most likely a combination of several of these factors interacting to alter testosterone concentrations.

The chronic effects of training on resting testosterone concentrations have not been conclusively proven. Wheeler et al. (1984) examined isolated, single blood samples in a comparative study and found significantly lower free and total testosterone

concentrations in chronically endurance-trained men at rest. Other studies support this finding (Hackney et al., 1998, 1990), reporting testosterone concentrations in trained subjects that were 69-77 % of the testosterone concentrations in untrained men. In studies that have collected and compared isolated blood samples over repeated days or weeks while exposing subjects to endurance training programs, the findings have been contradictory. Some studies have reported a significant reduction in resting testosterone occurs after up to 6 months of training (Wheeler et al., 1991), which are consistent findings with the studies that compared isolated, single blood samples. Other studies, however, have found no significant changes in resting testosterone concentrations after up to one year of training (Hakkinen et al., 1989). Rowbottom et al. (1997) observed a change in total testosterone, but not in the biologically active free testosterone serum concentrations over a nine-month training period in well-trained male triathletes. The conflicting results of these studies may be a function of the initial training status, and/or the magnitude of training administered and the volume of training load employed (Hackney, 1996).

2.3.2 Growth Hormone

Growth hormone (GH) is an anterior pituitary hormone produced by the somatotropic cells, or somatotropes. GH is an anabolic hormone that promotes protein synthesis and encourages the use of fats for fuel, thus conserving glucose (Marieb, 1995). Specifically, GH functions to 1) stimulate uptake of amino acids from the blood and incorporate them into cellular proteins; 2) mobilize fats from adipose tissue for transport to other cells, thereby increasing blood levels of fatty acids; and 3) decrease the rate of glucose uptake and metabolism (Kraemer et al., 1992; Marieb, 1995). GH promotes

protein synthesis by facilitating the transport of amino acids across the cell membrane and affecting transcription in the nucleus, resulting in increased amounts of RNA (Kraemer et al., 1992). In the liver, GH works to encourage glycogen breakdown and the release of glucose to the blood (Marieb, 1995).

Two hypothalamic hormones with antagonistic effects chiefly regulate the secretion of GH. Growth hormone-releasing hormone (GHRH) stimulates GH release, while growth hormone-inhibiting hormone (GHIH), also known as somatostatin, inhibits GH release. GHIH release is triggered by the feedback of GH and somatomedins. Increased levels of GH also create feedback to inhibit its own release. Secondary triggers such as stress, sleep patterns, exercise and nutritional factors may also influence GH release. Typically, GH secretion has a diurnal cycle with the highest levels occurring during nighttime sleep (Marieb, 1995).

During acute exercise, GH levels rise with a threshold level at approximately 30 % of maximal oxygen uptake (VO_2 max) (Jenkins, 1999). The extent of the rise in GH with acute exercise is dependent on the type, volume and intensity of the exercise, as well as the amount of muscle mass used (Felsing et al., 1992; Jenkins, 1999; Kraemer et al., 1992). Anaerobic exercise and hypoxia evoke the largest rise in GH with levels increasing up to 100-fold (Jenkins, 1999). Research has shown that women exhibit greater absolute GH secretion rates than men both at rest and during exercise, but exercise evokes a similar incremental GH response in both genders (Wideman et al., 1999).

In a study investigating exercise intensity and serum GH concentrations, Sutton and Lazarus (1976) exercised male subjects at 300, 600 and 900 $kpm \cdot min^{-1}$ for 20

minutes, representing 25-33 %, 40-66 % and 75-90 % of the subject's VO_2 max, respectively. The investigators observed a maximum elevation in serum GH concentrations following exercise at 900 kpm·min^{-1}, with a lessened response following exercise at 600 kpm·min^{-1}, and no effect on serum GH concentrations at the lowest exercise intensity. This demonstrates that the magnitude of the GH increase varies in proportion to the intensity of the exercise performed. The authors (Sutton & Lazarus, 1976) further noted that the peak elevation in GH occurs 5-15 minutes following the exercise stimulus.

Felsing and colleagues (1992) provide support for the work of Sutton and Lazarus (1976) in a study investigating the effects of duration and intensity of exercise on GH. The researchers exercised subjects at high and low intensities on a cycle ergometer for 1, 5 and 10 minutes at each intensity, for a total of 6 exercise sessions. The results demonstrated an increase in GH concentration following 5 and 10 minutes of high intensity exercise, although the increase after 5 minutes of high intensity exercise was not statistically significant. No increases in GH concentration were observed during the low intensity exercise at any duration, or after 1 minute of high intensity exercise. High intensity resistance training exercise has also been shown to result in a similar rise in GH concentrations (Kraemer et al., 1992; Vanhelder et al., 1984). The results of these studies indicate that the intensity, duration and mode of exercise all play roles in the GH response to exercise, a similar conclusion to that of other researchers (Kindermann et al., 1982).

However, the exact nature of training effects on the GH response is uncertain, given that researchers have reported different results. Bloom and colleagues (1976)

displayed a decreased response in GH in trained cyclists when compared with the same relative exercise intensity in untrained individuals, while other researchers demonstrated lessened GH responses to the same absolute exercise intensity after training (Weltman et al., 1997). Yet other research has observed increases (Weltman et al., 1992) or no change in GH with training (Bullen et al., 1984; Pitkanen et al., 2002). Thus, the effect of training on the GH response is not yet conclusive. Discrepancies between research findings may relate to differences in exercise duration and intensity during training; nutritional, hormonal and/or training status of the individual; individual adaptations to training; gender of the subjects; technical differences in obtaining hormonal measures (i.e. time of day) and the host of assay protocols.

2.3.3 Cortisol

Cortisol, also known as hydrocortisone, is the major glucocorticoid secreted by the zona fasciculata cells of the adrenal cortex (Marieb, 1995). The glucocorticoids influence the metabolism of most body cells and help to provide resistance to stressors. Under normal homeostatic conditions, the glucocorticoids help the body adapt to external changes and intermittent food intake by maintaining blood sugar levels at a fairly constant level (Marieb, 1995). Specifically, the principal functions of the glucocorticoids are concerned with the metabolism of carbohydrates, fats and proteins, and can be summarized as 1) stimulating gluconeogenesis; 2) mobilizing amino acids; and, 3) mobilizing fatty acids from adipose tissue (Marieb, 1995; Bunt, 1986). Glucocorticoids work to stimulate gluconeogenesis, which is the formation of glucose from noncarbohydrate molecules such as fats and proteins. Glucocorticoids increase the liver conversion of amino acids into glucose, thus increasing liver glycogen and blood glucose.

Glucocorticoids also mobilize amino acids from tissues and increase liver amino acids, resulting in a protein catabolic (breakdown) effect in muscle. Finally, glucocorticoids serve to mobilize fatty acids from adipose tissue, encouraging an increased use of fatty acids to meet the energy needs of most tissue cells, thus reserving glucose for the brain (Marieb, 1995; Tharp, 1975; Bunt, 1986).

Cortisol secretion is regulated by a typical negative feedback system. Cortisol release is promoted by adrenocorticotropic hormone (ACTH), also known as corticotropin, which is in turn stimulated by the hypothalamic releasing hormone corticotropin-releasing hormone (CRH). Increased cortisol levels act on both the hypothalamus and the anterior pituitary, inhibiting CRH release and turning off ACTH and cortisol secretion (Marieb, 1995). Cortisol secretory bursts, stimulated by patterns of eating and activity, occur in a definite pattern throughout a 24-hour period. Cortisol blood levels peak shortly after awakening in the morning, while the lowest levels are seen in the evening just before and shortly after sleep ensues (Marieb, 1995). The normal cortisol rhythm is interrupted by acute stress as the sympathetic nervous system overrides the inhibitory effects of elevated cortisol levels and triggers CRH release. The resulting increase in ACTH blood levels causes an outpouring of cortisol from the adrenal cortex. Stress results in a dramatic rise in blood levels of glucose, fatty acids and amino acids, all stimulated by cortisol (Marieb, 1995). The rise in blood levels of substrates due to cortisol may be beneficial to athletes and active individuals as there would be a greater provision of substrates to be used for energy, contributing to the ability of muscles to perform increased work (Tharp, 1975).

During acute exercise, changes in plasma cortisol concentrations are dependent upon the relative intensity of the exercise, the duration of the exercise and the fitness level of the participant (Jacks et al., 2002; Rudolph et al., 1998; Tharp, 1975). Rudolph and McAuley (1998) conducted a study in which they compared the salivary cortisol responses and ratings of perceived exertion (RPE) of runners and non-runners during and following a 30-minute treadmill run at 60 % VO_2 max. The researchers found that those participants who reported greater perceptions of effort during exercise demonstrated higher levels of cortisol 30 minutes post-exercise. This suggests that runners who perceived the exercise bout to be more physically demanding possessed increased cortisol levels post-exercise. There were no significant differences between groups in the cortisol levels during exercise, although both groups demonstrated an increase in cortisol levels from baseline to the 29th minute of exercise.

Luger and colleagues (1987) provide additional support for the contention that changes in plasma cortisol concentrations are dependent on the intensity of the exercise. The researchers compared the cortisol responses in sedentary, moderately trained and highly trained runners to graded levels of treadmill exercise (50, 70 and 90 % of maximal oxygen uptake). No elevation in plasma cortisol concentrations were observed in any of the groups at 50 % VO_2 max, while exercise intensities of 70 and 90 % VO_2 max were associated with a proportional increase in plasma cortisol concentrations. No differences were found in the cortisol response among the three groups of subjects at a given relative intensity, but it was noted that basal concentrations of plasma cortisol were significantly elevated in the highly trained subject at all three exercise tests compared to the sedentary and moderately trained subjects. Further, Luger et al. (1987) found that trained athletes

had an attenuated cortisol response when compared with untrained subjects at match absolute workloads. The changes in plasma cortisol concentration were inversely proportional to the level of physical training, suggesting that adaptation to regular aerobic exercise is associated with a reduced cortisol response.

Bunt (1986) suggests that while cortisol has been shown to increase during acute exercise, a delayed response exists and peak cortisol concentrations often occur after exercise. Cumming et al. (1986) support this conclusion. In a study investigating hormonal responses to a bout of acute exercise consisting of a graded exercise test to exhaustion (VO_2 max test), the researchers found that serum cortisol levels became significantly elevated by 30 minutes following the initiation of exercise, which was 11 to 15 minutes after the termination of the exercise test (Cumming et al., 1986).

Research conducted by Neary et al. (1994) to examine the effects of training on urinary free cortisol as an indicator of exercise training stress, suggests that cortisol levels are significantly increased with training and remain elevated until after training. Neary and colleagues (1994) trained 27 experienced male cyclists 4 times per week over 7 weeks, measuring urinary free cortisol on a weekly basis. Urinary free cortisol levels in sedentary control subjects were significantly lower than pre-training values in the cyclists, suggesting that fitness of the individual plays a role in the cortisol response to stressors.

In a study on cortisol and growth hormone levels in the course of further improvement of performance capacity in trained rowers, Snegovskaya and Viru (1993) also found that the fitness of the individual plays a role in cortisol concentrations. The researchers observed that pre-competition concentrations of cortisol were significantly

higher in elite rowers than in rowers of lower performance levels. In addition, Snegovskaya and Viru (1993) observed that further improvement of performance capacity in rowers was associated with increased cortisol levels, suggesting that cortisol levels rise with training. This is in agreement with the work of Hakkinen et al. (1989), in which increased serum cortisol concentrations were observed in elite endurance trained swimmers over the course of one year.

2.4 Research Problems and Concerns when Investigating Hormones

The examination of studies investigating hormone concentrations during acute maximal and submaximal exercise, as well as chronic conditions such as training, reveals several potential methodological issues that exist that make it difficult when interpreting the research findings (Hackney, 1996). First, different research design concerns may influence outcomes. One method of investigating hormonal research questions involves taking single, isolated blood samples from different subjects and comparing them in a retrospective, comparative study. Another method involves taking a series of blood samples from the same subject, often throughout the course of a training program, in a prospective study.

Retrospective studies have limitations with regards to research involving exercise training in that they are often compare untrained subjects to trained subjects. The subjects that are considered to be trained have exposed themselves to exercise for a period of time (usually years), and the investigator is observing the final product. It is not taken into consideration that the subjects may have employed a variety of training methods and may have varying individual responses to the intensity, volume and type of training, all of which may affect each subject's hormonal response to training.

Prospective studies are often difficult to compare as individual investigators define training differently and have varying definitions of what is considered to be a 'trained state'. In order to appropriately compare endurance training studies, it is important to ensure that the hormone samples were taken when the subject was in a steady state of training; that is, there were no drastic changes (i.e., increased volume and/or intensity) in the training regime such as might be experienced when preparing for a competition. This will enable better comparison of hormone measures during serial sampling (Hackney, 1996).

Another limitation to research studies involving hormones and exercise is the assumption that any observed changes in hormones are a result of the exercise program. This assumption may not be completely true unless all other factors that may affect hormones are controlled for, including, but not limited to, diet, emotional stress, genetics, sleep loss and weight loss (Bunt, 1986; Hackney, 1996). Controlling for all of the external factors that may affect hormone concentrations would be very difficult to do, if not impossible.

A further factor that may limit the comparison of research studies investigating hormones is the time of day blood measures are taken (Jenkins, 1999). Many hormones, including testosterone, growth hormone and cortisol, have pulsatile releases that will influence the concentration of the hormone in the blood, depending on what time of day the measure is taken. This must be taken into account when interpreting study results.

Finally, it must be noted that observed increases in serum levels of a hormone during exercise do not necessarily reflect an increase in secretion of the hormone (Bunt, 1986). Increases may be as a result of decreased metabolic clearance or decreases in

plasma volume leading to an increased haemoconcentration of the hormone (Bunt, 1986). Shepard and Sidney (1975) support this hypothesis, noting that an increase of plasma hormone concentration may reflect (1) an increase in the rate of secretion, (2) a decrease in excretion or metabolism, (3) release from a binding site, and, (4) haemoconcentration. Thus, until further research is conducted, it cannot be decisively concluded that an increase in serum hormone concentration is necessarily a result of increased hormone secretion.

2.5 Elk Velvet Antler

2.5.1 Introduction

Velvet antler has been used in traditional Oriental medicine for thousands of years as a supplement to aid with arthritis, anaemia, and fertility and as an aphrodisiac (Batchelder, 1999). Velvet antler is regarded in China as preventive medicine, taken as a tonic to maintain good health and cure sickness (Sunwoo et al., 1995). It is yang (positive) in property, helping to restore harmony between the conflicting yin (negative) forces, in order to secure health (Batchelder, 1999).

Due to an increasing demand for velvet antler, deer farming is a rapidly growing industry in many parts of the world, including North America, Europe and New Zealand (Sunwoo et al., 1995). More than 1000 tons of velvet antler are produced yearly, with New Zealand and China being the larges producers (Batchelder, 1999). In 1997, Alberta alone reported 307 deer farms with 5,267 head of livestock and a total production potential of 52,102 lbs (23,642 kg) (Alberta Agriculture, 1999).

Velvet antler may come from a variety of species of deer, including spotted deer (*Cervus nippon hortulorus*), maral deer (*Cervus elaphus sibiricus*), and Canadian deer or

wapiti (*Cervus elaphus xanthopygus*). Antlers grow in length by endochondral ossification and in diameter by intramembranous ossification. The antlers are removed at approximately 60 days of growth, or approximately 65 days after casting of the buttons from the previous set (Sunwoo et al., 1995). Antlers are harvested under general anaesthesia of the animal, cutting a minimum of 1.5 cm above the top of the pedicle using a clean and sharp saw (The Deer and Elk Procedures Manual, 2000). The antler itself can be divided into four different sections (tip, upper, middle and base), each of which has a varying chemical composition (Sunwoo et al., 1995). The four sections are often combined when producing supplements for commercial sale. Once harvested, the antler is dried prior to its manufacture into various medicinal forms. This may be done through preservation, freeze-drying or food-drying methods.

2.5.2 Elk Velvet Antler Composition

Velvet antler is composed of protein, collagen, lipid, uronic acid, sulfated glycosaminoglycan (GAG), sialic acid, ash, calcium, phosphorus and magnesium, with varying amounts of each in the different sections of antler (Sunwoo et al., 1995). Protein, collagen, ash, calcium, phosphorus and magnesium all increase downward, while the lipids, uronic acid, and sulfated GAG are more concentrated in the tip and upper sections of the antler (Sunwoo et al., 1995).

Sunwoo et al. (1995) calculated the whole antler to be 31 % dry matter, 57 % protein and 39 % ash. Collagen has been suggested to be the major protein in the antler, with the amount of collagen in dry matter increasing downward in the antler. The major function of collagen is to provide mechanical strength to the tissue, thus it would make

sense that there would be increased amounts of collagen lower in the antler to support the distal tissue (Sunwoo et al., 1995).

2.5.3 Recommended Doses of Elk Velvet Antler

There is a paucity of research existing on velvet antler as a supplement, thus there are no recommended doses confirmed in the literature on using velvet antler as a supplement to athletic performance. Although no dose-response research has been conducted on velvet antler, there are varying doses of velvet antler recommended by the supplement manufacturers. Royal Elk Products of Sangudo, Alberta (the manufacturer of the elk velvet antler used in the present study) recommend a dose of two tablets, each containing 280 mg EVA, per day, for a daily total of 560 mg. Planetary Formulas Antler Velvet comes in 250 mg tablets, with a recommended dosage of two tablets, one to two times per day, for a total of 500–1000 mg daily (http://www.iherb.com/antlervelvet.html). Qeva Elk Velvet Antler Products recommends three 280 mg capsules daily for the first two weeks, then one to two capsules per day following that (http://www.qeva.com/products/people.htm). This equates to a daily total of 840 mg for the first two weeks, then 280-560 mg daily for subsequent doses. Velvita recommends an initial dose of 1500 mg per day for an initial loading phase of two weeks, then 500-1000 mg per day following that (http://sexualboost.com/faq.htm). Unpublished research supported by Impax Therapy (http://impaxsupply.com/sports.htm) quotes separate studies that have supplemented athletes with low doses (350 mg) and high doses (12 gm) deer velvet antler. Top New Zealand athletes such as Hamish Carter (triathlon) and Rhys Duggan (rugby) report positive effects on performance with trialing 900 to 1200 mg of velvet antler per day. Thus, while no dose-response relationship has been established for

velvet antler supplementation, a wide variety of doses are recommended by manufacturers and ingested by people worldwide.

2.6 Velvet Antler and Exercise

There is a deficiency of research in the literature examining the effects of EVA supplementation on exercise and training. Sleivert and colleagues (in press) conducted a study in New Zealand examining the effects of deer antler velvet supplementation on aerobic power, erythropoiesis and muscular strength and endurance characteristics. Thirty-eight subjects were randomly divided into three groups, deer velvet extract (n=12), powder (n=13) or placebo (n=13). Subjects strength trained three times per week for 10 weeks, while daily ingesting deer velvet extract (300 mg/day), deer antler velvet powder (1.5g/day), or a placebo powder. All subjects were measured for circulating levels of testosterone, insulin-like growth factor, erythropoietin, red cell mass, plasma volume and total blood volume. Physiological performance testing included determinations of VO_2 max and muscular strength and endurance. All groups improved 6 RM strength equivalently, but the powder group demonstrated a greater increase in isokinetic knee extensor strength and endurance when compared to the placebo group. No group experienced endocrine, red cell mass or VO_2 max changes.

While little research has been published investigating EVA supplementation in human subjects, one study has reported increased plasma testosterone concentrations following repeated administration of deer antler extract in senescence-accelerated mice (Wang et al., 1988). A second study, conducted by Clifford and colleagues (1979) examined the effects of velvet antler extract on cardiovascular dynamics in dogs and noted an elevation in stroke volume during the period with which the dogs were

administered velvet antler extract. Thus, it appears that velvet antler supplementation may have benefits to muscular strength and endurance, as well as aerobic power. However, more research must be conducted before any potential ergogenic benefits can be deemed conclusive.

2.7 Conclusion

Although the research is not conclusive, it appears that many training regimes that simultaneously train both strength and endurance may create an "interference effect" that compromises the development of strength. This could have detrimental effects to elite athletes in sports such as rowing that demand high levels of both strength and endurance to be successful. Therefore, further strategies must be found to avoid this potential interference effect in strength development and enable athletes performing concurrent training to optimize all the training benefits. Velvet antler, used for centuries in traditional Oriental medicine as a tonic to maintain good health and cure sickness, may be such a strategy to help avoid an interference effect with concurrent training. Several world-class athletes have reported positive effects to training while supplementing with EVA. If velvet antler is able to create an enhanced anabolic environment while training, this supplement may help individuals to overcome any interference effect incurred as a result of concurrent training. Further research is needed to investigate the effects of velvet antler supplementation on training.

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CHAPTER 3

Effect of Elk Velvet Antler Supplementation During Concurrent Training in Rowers

3.1 Introduction

Rowing is a sport that demands high levels of both strength and endurance to be successful (Secher, 1993). The physiological basis to perform the sport is primarily developed through strength and aerobic endurance training. However, some researchers have suggested that an "interference effect" in strength development may occur as a result of simultaneous strength and endurance training (Bell et al., 1997; Bell et al., 2000; Dudley & Djamil, 1985; Hennessey & Watson; 1994; Hickson, 1980; Kraemer et al., 1995), such that the effectiveness of the strength training is compromised.

The mechanisms behind the interference effect of simultaneous strength and endurance training are not completely known. Leveritt and colleagues (1999) propose an acute hypothesis for the interference effect, contending that residual fatigue from the endurance training component compromises the ability to develop force or tension during the strength element of concurrent training, thereby preventing optimal strength development and adaptation from occurring. Some researchers (Dudley & Fleck, 1987; Hickson, 1980) postulate that the impairment of strength development, but not to endurance, during concurrent training may be directly related to the muscles inability to adapt to both forms of exercise, as the adaptations to strength and endurance training are divergent in nature. Research by Bell et al. (2000) supports the contention that combined strength and endurance training can suppress some of the adaptations to strength training and augment some aspects of capillarization in skeletal muscle, which is an adaptation to

endurance training. Other research by Bell et al. (1991) suggests that the interference of strength development is due to a lack of muscle fibre hypertrophy. A further explanation for the interference effect on strength development during concurrent strength and endurance training suggests that concurrent training may hinder organization of efficient motor unit recruitment patterns necessary for forceful muscular contractions (Chromiak & Mulvaney, 1990; Dudley & Djamil, 1985).

A final explanation for the interference effect may be related to an increase in cortisol as a result of training (Bell et al., 1997; Bell et al., 2000; Kraemer et al., 1995, Leveritt et al., 1999), which has been demonstrated to inhibit testosterone release (Cumming, 1987). Anabolic hormones such as testosterone and growth hormone are critical in maintaining and building skeletal muscle mass through strength training (Bell et al., 2000; Kraemer et al., 1995). Thus, if anabolic hormones are inhibited, strength development may be compromised. Therefore, further strategies must be found to avoid a potential interference effect in strength development for athletes performing concurrent strength and endurance training and enable them to optimize the training benefits.

Athletes and commercial supplement companies have made many anecdotal claims about the effects of velvet antler that may work to counteract the interference of strength development with concurrent training. Velvet antler has been said to aid performance, boost stamina, strength and efficiency, as well as work to increase muscle mass. Velvet antler has been used as a traditional medicine in the Orient for thousands of years, for ailments such as arthritis and infertility; however, the mechanisms by which it may aid athletic performance are not known. Velvet antler from Wapiti (*Cervus elaphus*) have been shown to contain a variety of amino acids, lipids, ash, collagen, calcium,

phosphorus, magnesium, sialic acid, uronic acid and sulfated glycosaminoglycans (GAGs) (Sunwoo et al., 1995). It is also thought that elk velvet antler (EVA) may contain hormones, prostoglandins and growth factors, possibly providing steroid hormone precursors that contribute to an anabolic hormone environment. A paucity of peer-reviewed literature exists to identify the effect velvet antler may have on hormones and athletic performance.

The purpose of this study is to examine the effects of EVA supplementation on hormones and various measures of rowing performance and physiological parameters after 5 and 10 weeks of combined resistance and endurance training. It is hypothesized that subjects who supplement with EVA while training will have enhanced performance over subjects training with a similar program, but ingesting a placebo. As a result, subjects who supplement with EVA will have an enhanced anabolic profile when compared to the placebo group, enabling them to maintain strength development while performing concurrent strength and endurance training.

3.2 Subjects and Methods

3.2.1 Subjects and Experimental Design

A total of 46 male and female volunteers from the University of Alberta and the Edmonton rowing community were randomly assigned by gender into one of two groups: elk velvet antler supplementation (EVA) (12 males, 9 females) or placebo (PL) (13 males, 12 females). The mean ± standard deviation for age, height and initial body mass of all subjects was 25.3 ± 5.3 years, 174.3 ± 8.2 cm, and 73.0 ± 11.3 kg, respectively. One female subject was randomized into the EVA group at baseline, but did not complete all of the physiological testing due to a back injury sustained during activities outside the

scope of the study. The volunteers were primarily physically active university students and all had had some experience with strength and endurance training. All subjects completed an informed consent and PAR-Q prior to participation. The study was reviewed and approved by the Research Ethics Committee of the Faculty of Physical Education and Recreation at the University of Alberta.

The study was a randomized, double blind, placebo-controlled, pre-, mid- and post-test design. The experimental design included 10 consecutive weeks of training for both groups. All subjects performed identical regimes of strength and endurance training on alternate days (6 days per week). All physiological testing was conducted before and after 5 and 10 weeks of training (see Figure 1) in the same order at each testing period. All subjects were asked to refrain from any exercise outside of the structured training program for the duration of the study. Subjects in the EVA group consumed one 280 mg velvet antler capsule, twice per day for the 10-week training period, for a daily total of 560 mg. Subjects in the PL group consumed an equal amount of gelatin capsules containing whole-wheat flour. Both the velvet antler and placebo were contained in identical bottles, with an independent lab technician ensuring each subject received the correct bottle.

3.2.2 Physiological Tests

Body mass and height were recorded using a Health-o-Meter scale (Sunbeam Products, Purvis, MS) and a wall mounted tape measure with a set square. Percent body fat was obtained from 6 skinfold sites (triceps, subscapular, iliac crest, front thigh, chest, and abdomen (left) – females substitute rear thigh for chest) using the Yuhasz calculation (PFLC Resource Manual, 1996) and Harpenden skinfold calipers (UK). Subjects

performed, in order, a 2000 m timed rowing test, a maximal oxygen consumption (VO_2 max) test and mRM leg and bench press strength tests during each testing period. All tests were separated by a minimum of 24 hours, with the exception of height, body mass and skinfold measurements, which were performed immediately prior to the VO_2 max test.

The 2000 m rowing performance test involved a simulated rowing time trial in which the subjects completed 2000 m as fast as possible after a standardized warm-up. Total elapsed and 200 m interval split times, power output, stroke rates and heart rates were recorded during and at the completion of the test.

To determine absolute upper and lower body strength measures, all subjects underwent a multiple repetition maximum (mRM) bilateral incline leg (LP) and bench press (BP) test, as previously reported (Syrotuik, 2001).

Maximal oxygen consumption (VO_2 max) was determined by using a continuous incremental protocol to exhaustion on a Concept IIC rowing ergometer (Morrisville, VT). Female subjects began rowing at 50 W, while male subjects began at 100 W. Power output was increased by 50 W every two minutes until the subject could no longer maintain the required power output and had a respiratory exchange ratio (RER) > 1.0. At this point, the subject was instructed to go "all out", or as hard as they could, until volitional exhaustion occurred. VO_2 max was indicated by a peak and plateau (< 100 ml/min change) in VO_2 with continued exercise. Additional supportive criteria for the confirmation of VO_2 max included: RER > 1.1; heart rate at or above age predicted maximum; and, volitional exhaustion. During the test, expired gases were collected and analyzed using a Medgraphics CPX/D Metabolic Cart (Medical Graphics, St. Paul, MN)

that was calibrated before and after each test with known gas concentrations. The VT determined during the VO_2 max test was used to prescribe the intensity of the endurance training. The subject's heart rate was continuously monitored using a Polar Pacer HR telemetry heart rate monitor system (Polar Canada, PQ). A complete description of the physiological testing procedures can be found in Appendix A.

3.2.3 Blood Procedures

A registered nurse drew blood at rest, and 5 minutes and 60 minutes after completion of the 2000 m rowing test to determine total serum concentrations of free and total testosterone, human growth hormone, and cortisol. Resting blood samples were obtained between 3 and 6 pm on the day prior to the 2000 m rowing test, after 24-48 hours of no training. The late afternoon sampling was considered to be a period of relative stability in serum testosterone, when circulating levels are low during the 24-hour cycle (Cumming, 1987). The venous blood samples were obtained from a superficial arm vein using a needle, syringe and vacutainer assembly. Whole blood was processed and serum samples were stored at in a freezer at -80°C until analyses were performed. For serum, whole blood was allowed to clot at room temperature for 15 to 20 minutes and was then centrifuged at 3000 g for 10 min. Samples were then aliquoted into separate analysis tubes and frozen. Serum was subsequently analyzed in duplicate, using commercially available ^{125}I radioimmunoassay kits for serum free testosterone, human growth hormone (DiaSorin, Stillwater, MN), total testosterone, and cortisol (Diagnostic Products Corporation, Los Angeles, CA). Samples were thawed only once and decoded after all analyses were completed (blinded procedure).

3.2.4 Physical Training Program

Subjects completed a 10-week training program, consisting of three strength and three endurance training sessions per week. Strength training consisted of 4 core (bench press, lat pulldowns, seated rows, and bilateral incline leg press) and 6 supplemental (upright rows, bicep curls, triceps pushdowns, knee flexion, knee extension, and calf raises) exercises. Initial training loads were established from an mRM strength assessment on each exercise, as previously noted. Volume and intensity were progressively overloaded using a periodized program with the aid of a computer software program (B. E. Software, Lincoln, NE). Core lifts were performed at 70-95 % of predicted 1RM, 2-10 repetitions for 4-5 sets while supplementary lifts were executed using ranges of 65-85 % of predicted 1RM, 8-10 repetitions for 2-3 sets.

Endurance training consisted of 2 continuous and 1 interval training sessions per week on a Concept II rowing machine, completed on opposite days to resistance training. The first continuous training session each week was set at an intensity equivalent to a 5 second split time range just below the split time/power output at ventilatory threshold and adjusted at halfway according to the combined VO_2 max/ventilatory threshold testing results. The second continuous training session each week was set at an intensity equivalent to a 5 second split time range just above ventilatory threshold and adjusted at halfway according to physiological testing results. The interval training sessions were set at an intensity equivalent to the mean split time that the 2000 m simulated rowing test was performed at. All subjects wore HR monitors (Polar Canada, QC) during the endurance training; the sessions were supervised and training logs kept. The weekly

volume of endurance training increased every two weeks, beginning at 20,000 m and increasing to 27,500 m after 10 weeks.

3.2.5 Statistical Analysis

Results for performance data were analyzed using a 2 X 2 X 3 ANOVA (gender · group · time), which involved a comparison between groups (EVA and PL) over the course of training (repeated measures). Results for hormone data were analyzed using a 2 X 2 X 3 X 3 ANOVA (gender · group · exercise · training) with repeated measures on the last two variables. Post hoc comparisons were performed on significant F ratios using a Newman Keuls test. Results were considered significant at the $p < 0.05$ level for all analyses. The statistical software program used was Statistica (1999 Edition, Oklahoma City, OK, USA). All data are means ± SD unless otherwise noted.

3.3 Results

Effect of training and EVA supplementation on the physical fitness parameters:

The EVA subjects had a mean age of 26.3 ± 5.8 years and height of 173.6 ± 8.6 cm. The PL group had a mean age of 24.1 ± 4.6 years and height of 175.1 ± 7.8 cm. There was no significant difference between the body mass scores or the sum of skinfolds between EVA and PL groups, but there was a significant difference between the body mass scores and the sum of skinfolds of males and females (Table 1). As well, there was a significant difference in the main effect of sum of skinfolds between before and after 10 weeks of concurrent strength and endurance training for all groups, indicating that all groups decreased their total subcutaneous body fat in the skinfold sites measured over the course of the training period. No significant changes were found for body mass scores between before and after 5 and 10 weeks of training.

There was a significant increase from baseline in both relative and absolute VO_2 max scores (Table 2) after 5 and 10 weeks of training. No significant differences were observed between the PL and EVA groups, although males had significantly higher relative and absolute VO_2 max scores than females at all time periods.

Males and females in both the EVA and PL groups significantly increased predicted bench and leg press strength from baseline scores after 5 and 10 weeks of training (Table 3). Significantly higher predicted bench press and leg press strength scores were recorded in males over females in both the EVA and PL groups at all time periods. No interaction effects were observed between EVA and PL groups.

Table 4 shows the effects of EVA and placebo supplementation on 2000 m rowing time before and after 5 and 10 weeks of concurrent strength and endurance training. Males had significantly lower 2000 m rowing times than females. All groups demonstrated significantly lower 2000 m rowing times from baseline after 5 and 10 weeks of training. As a result of randomization, the EVA group had a significantly lower 2000 m time at baseline than the PL, as well as after 5 and 10 weeks of training. No interaction effects were observed between groups and 2000 m rowing time, and the EVA group did not decrease their rowing time by a significantly larger margin than the PL group.

Effect of training and EVA supplementation on serum hormone concentrations:

Tables 5 and 6 present data that shows the effects of EVA and PL supplementation on serum hormone concentrations at rest, 5 and 60 minutes after a 2000 m simulated rowing race, as well as prior to and after 5 and 10 weeks of concurrent strength and endurance training in males and females. Serum hormone concentrations of

total testosterone and free testosterone were significantly different between males and females. No training effect was found for total or free testosterone, in either males or females, although males were found to have significantly increased total and free testosterone levels at 5 minutes post exercise when compared to baseline and 60 minutes post exercise values (figures 2 and 3). No differences were observed between baseline and 60 minutes post exercise values. No significant differences were found between the EVA and PL groups (figure 4).

No training effect was observed for either males or females in relation to serum growth hormone concentrations. Males had significantly different serum growth hormone concentrations from females at 5 and 60 minutes post exercise, but not at rest. At 5 minutes post-exercise, female serum growth hormone concentrations were higher than males, but by 60 minutes, male values were higher than females (figure 5). No differences were found between the EVA and PL groups.

Serum cortisol concentrations demonstrated a main effect for exercise (figure 6). Concentrations at 5 and 60 minutes post exercise were significantly increased over baseline concentrations, but were not different from each other regardless of gender or group. Serum cortisol concentrations were also found to have an interaction effect for exercise and training (figure 7). No differences were found in baseline values at any testing period. Serum cortisol concentrations at 5 minutes post exercise after 5 weeks and 10 weeks of training were significantly higher than pre-training values, but not from each other. 60 minutes post exercise serum cortisol concentrations were significantly increased after 10 weeks of training when compared to the pre-training and after 5 weeks of training values. Pre-training recovery values (60 minutes post exercise) were not

significantly different from those after 5 weeks of training. Thus, 5 weeks of training made the cortisol response increase significantly immediately after exercise, but did not keep the response elevated at 60 minutes post exercise. Ten weeks of training increased the cortisol response immediately after exercise and maintained the elevated response at 60 minutes post exercise.

3.4 Discussion

Some research has pointed to an "interference effect" in some aspects of strength development while training both strength and endurance simultaneously (Bell et al., 1997; Bell et al., 2000; Dudley & Djamil, 1985; Hennessy & Watson, 1994; Hickson, 1980; Kraemer et al., 1995). Other research does not support these findings (McCarthy et al., 1995; Nelson et al., 1990; Sale et al., 1990). The reasons for a compromise in strength development are unknown, however, some research findings speculate that the interference effect may be related to an increase in cortisol due to training (Bell et al., 2000; Kraemer et al., 1995), resulting in an attenuated anabolic environment. If anabolic hormones are inhibited, strength development may be compromised.

The aim of the present study was to investigate the effects of EVA supplementation on various physiological measures of rowing performance during 10 weeks of combined strength and rowing (aerobic) training. It was hypothesized that subjects who supplemented with EVA while training would have enhanced performance over subjects training with a similar program, but ingesting a placebo. As a result, subjects who supplemented with EVA would have an enhanced anabolic profile when compared to the placebo group, enabling them to maintain strength development while performing concurrent strength and endurance training.

Three main findings emerged from this study:

1. The 10 week training program improved all physiological measures of rowing performance that were tested, in all groups.
2. Serum testosterone, cortisol and growth hormone concentrations indicated that the EVA group did not have an enhanced anabolic environment when compared to the PL group.
3. It appears that 10 weeks of EVA supplementation coupled with training does not significantly improve rowing performance greater than training alone.

Both the EVA and PL groups, male and female, achieved similar results from the training program. All groups significantly decreased sum of skinfold scores after 10 weeks of training, and increased predicted leg press strength, predicted bench press strength, and both relative and absolute VO_2 max scores after 5 and 10 weeks of concurrent strength and endurance training. In addition, all groups significantly decreased 2000 m simulated race rowing time from baseline after 5 and 10 weeks of training. These results can be expected from the training program and are consistent with other research examining the effects of concurrent strength and endurance training (Bell et al., 1997; Bell et al., 2000; Hennessey & Watson, 1994; Hickson, 1980).

The present study demonstrated an enhanced catabolic environment during the recovery period following the 2000 m test in all groups, as was evidenced by the elevated serum cortisol response immediately after exercise, which remained elevated at 60 minutes post exercise, following 10 weeks of concurrent strength and endurance training. After only five weeks of training, the serum cortisol concentration was elevated at 5

minutes post exercise, but did not remain elevated at 60 minutes post exercise. This suggests that 5 weeks of concurrent strength and endurance training was not enough to create an enhanced catabolic environment during the recovery phase.

No training effect was found for total or free testosterone, in either group or gender, although males were found to have significantly increased serum total and free testosterone concentrations at 5 minutes post exercise when compared to baseline and 60 minutes post exercise values. These results support the work of Bell et al. (1997), who examined the effect of strength training and concurrent strength and endurance training on strength, testosterone and cortisol. Bell and colleagues measured serum hormone concentrations over the course of a 16-week training period, and observed no significant changes in total testosterone in either gender or in the strength or concurrent training groups, over the training period. The present study also demonstrated significantly increased serum testosterone concentrations following a short-term, intense bout of exercise (2000 m rowing test), which further lends support to previous research (Cumming et al., 1986; Galbo et al., 1977). No training effect was observed for either males or females in relation to serum growth hormone concentrations (Pitkanen et al., 2002) in the present study.

No interference to strength development occurred in the present study. This is evidenced in Table 3, which demonstrates that all groups displayed a statistically significant linear response in both leg and bench press strength development from baseline through 5 and 10 weeks of training. Whether the absolute strength gains for bench and incline leg press would be greater following 10 weeks of resistance training only versus the combined strength and endurance design of the present study is difficult

to determine since the present study did not employ an strength only experimental group in its design.

The results of the serum hormone concentrations suggest that 10 weeks of concurrent strength and endurance training does not induce an increased anabolic state, with or without EVA supplementation, which is critical in maintaining and building skeletal muscle mass as a result of strength training (Bell et al., 2000; Kraemer et al., 1995). This may be due to an increase in cortisol as a result of training (Bell et al., 1997; Bell et al., 2000; Kraemer et al., 1995).

Several world-class athletes have reported positive training effects in the popular media while supplementing with EVA. The components of velvet antler include: protein, collagen, lipid, uronic acid, sulfated glycosaminoglycan (GAG), sialic acid, ash, calcium, phosphorus and magnesium (Sunwoo et al., 1995). It is also thought that EVA may contain hormones, prostoglandins and growth factors, possibly providing steroid hormone precursors that contribute to an anabolic hormone environment. The mechanism of action of EVA to enhance athletic performance is not known.

Little research has been published in the literature examining the effects of velvet antler supplementation on athletic performance. One study, conducted by Clifford and colleagues (1979) examined the effects of velvet antler extract on cardiovascular dynamics in dogs and noted an elevation in stroke volume during the period with which the dogs were administered velvet antler extract. A second study has reported increased plasma testosterone concentrations following repeated administration of deer antler extract in senescence-accelerated mice (Wang et al., 1988). None of the animals in the above-mentioned studies underwent any form of exercise training during the study.

Research by Sleivert et al. (in press) examined velvet antler supplementation in humans while completing a strength training program and found that both the deer antler and placebo supplements groups significantly increased 6RM squat strength at each testing session over the 10-week training period, with no significant differences in the extent of change between groups. The researchers did discover, however, a significantly greater increase in isokinetic knee strength and endurance in the group supplementing with deer velvet powder over the placebo group. No differences between groups were found in total testosterone levels either pre- or post-training, which is in agreement with the results of the present study. This suggests that the increase in strength gains in the powder group was not as a result of an up-regulation of protein synthesis caused by elevated levels of testosterone, nor did the deer velvet antler supplementation result in an enhanced anabolic environment during the course of the study. The reasons for the significantly greater increase in isokinetic knee strength and muscular endurance are not known. It is possible though, that the deer velvet powder group displayed increased physiological adaptations to the training stimulus over the other groups as they had lower isokinetic strength levels in comparison to other groups at the start of the training program. It well known that, when exposed to the same training stimulus, individuals with lower levels of physiological function will adapt to a greater extent than individuals starting at a higher level of physiological function (Bouchard et al., 1988).

A second possible explanation of the increased muscular strength and endurance in the deer velvet powder group in Sleivert's study (in press) is in the processing methods used to create the powder and extract forms of deer velvet. The processing method used to create the powder form of deer velvet could have influenced the "active" properties of

the supplement, resulting in greater training effects. Additional research is needed to establish the role of the processing method of deer velvet on physiological adaptations to training before the effects can be completely understood. Further, the fact that the velvet powder group consumed 1.5 g/day of the supplement compared to 300 mg/day in the velvet extract group may have played a role in the results. A study examining the dose-response relationship of deer velvet on physiological measures is needed to better understand the role dosage played in Sleivert's study. Finally, it is possible that the effects of deer velvet powder are particular to the measurement criterion involved. This may explain why the velvet powder group demonstrated significantly increased knee strength and endurance when measured using an isokinetic dynamometer, but not when tested using a 6RM parallel squat test.

3.5 Future Research

Future research is needed in this area before the full effects of EVA as an ergogenic aid are understood. The present study examined the effect of EVA on the impairment of strength during concurrent strength and endurance training in rowers. It is possible that EVA may alter cardiovascular dynamics and enhance endurance performance (Clifford et al., 1979). These physiological variables were not considered in the present study. Further, research indicates that EVA may have potential to increase muscular strength and endurance in athletes using a strength training program (Sleivert et al., in press). In addition, research is needed to examine the dose-response relationship of EVA. As no dosage guidelines have been clinically established, we chose to follow the manufacturers recommendations for dosage at 560 mg/day. It is possible that this dosage was not enough to elicit a response. Finally, further research is warranted into the effects

of EVA on trained and untrained populations. The population for the present study involved a physically active sample of men and women already at an elevated level of strength and rowing performance. The capacity to improve, and thus to be affected by the supplement, may be greater in a group of less experienced or more sedentary subjects.

3.6 Summary

The results show that EVA supplementation coupled with training did not significantly improve any of the training indices over training alone, nor did it create an enhanced cellular anabolic environment. Thus, the data from the present study do not support an ergogenic effect for rowing performance or other training performance indices measured in this study, when ingesting EVA combined with concurrent strength and endurance training over a 10-week period in club level rowers. Additional research is needed to further examine the potential of EVA as a supplement for athletic performance enhancement and understand its mechanisms of action before EVA should be recommended as an ergogenic aid.

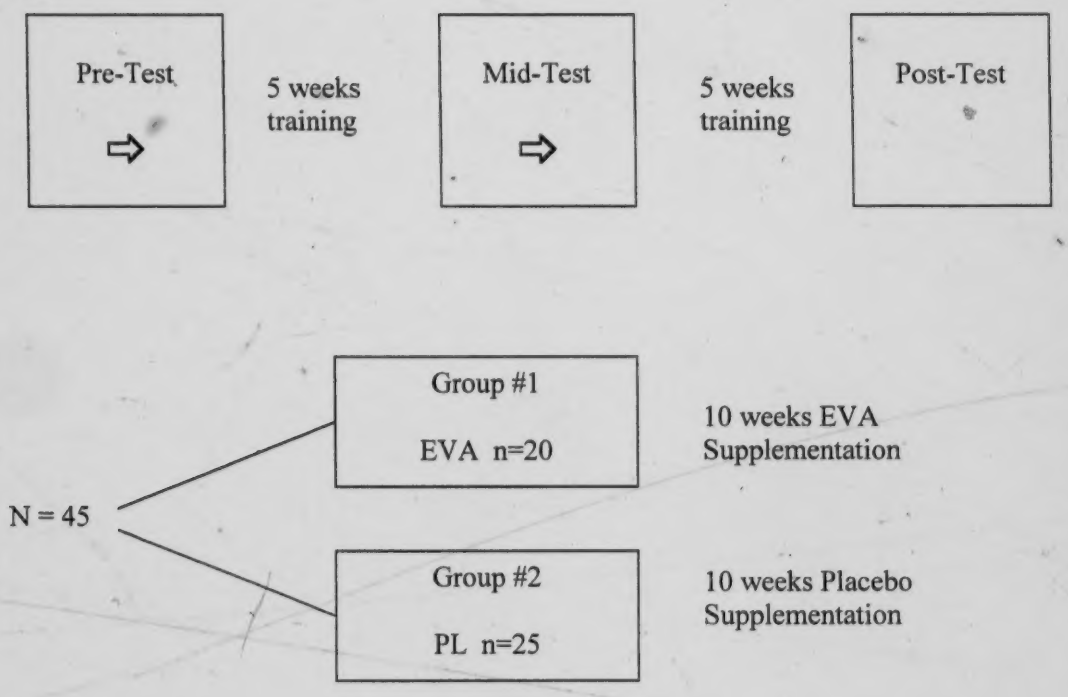

Figure 1: Experimental Design

Table 1. The effects of elk velvet antler (EVA) and placebo (PL) supplementation on body mass, % body fat and sum of skinfolds before and after 5 and 10 weeks of concurrent strength and endurance training. Values are means ± standard deviations.

		Training	Body mass (kg)	% Body Fat	Sum of Skinfolds (mm)
Male	**EVA**	Before	76.0 ± 7.1*	11.3 ± 5.0*	75.1 ± 41.2*
		5 weeks	76.4 ± 6.6*		
		10 weeks	76.0 ± 6.3*	10.6 ± 4.3*#	68.4 ± 34.3*#
	PL	Before	79.1 ± 10.6*	11.6 ± 4.6*	78.4 ± 43.5*
		5 weeks	79.1 ± 10.1*		
		10 weeks	78.4 ± 9.2*	10.9 ± 4.4*#	71.4 ± 40.9*#
Female	**EVA**	Before	70.2 ± 12.2	27.7 ± 11.2	148.0 ± 51.5
		5 weeks	71.3 ± 12.0		
		10 weeks	70.8 ± 11.7	26.6 ± 11.0#	143.3 ± 50.8#
	PL	Before	65.3 ± 11.4	23.2 ± 9.4	125.7 ± 44.3
		5 weeks	65.6 ± 10.8		
		10 weeks	66.3 ± 11.7	22.2 ± 8.1#	121.2 ± 38.7#

kg = kilogram, mm = millimetres

*Denotes males significantly different from females, $p<0.05$. # Denotes significantly different from before training, $p<0.05$. % Body fat and sum of skinfold measures were not taken after 5 weeks of training as it was not felt differences would be seen in such a short time period.

Table 2. The effects of elk velvet antler (EVA) and placebo (PL) supplementation on VO_2 max before and after 5 and 10 weeks of concurrent strength and endurance training. Values are means ± standard deviations.

		Training	Absolute VO_2 max ($L \cdot min^{-1}$)	Relative VO_2 max ($ml \cdot kg^{-1} \cdot min^{-1}$)
Male	**EVA**	Before	3.5 ± 0.4*	46.4 ± 5.8*
		5 weeks	3.9 ± 0.3*#	51.2 ± 4.0*#
		10 weeks	4.2 ± 0.5*##	55.0 ± 4.0*##
	PL	Before	3.7 ± 0.5*	47.3 ± 8.3*
		5 weeks	4.1 ± 0.4*#	52.1 ± 6.9*#
		10 weeks	4.4 ± 0.4*##	56.7 ± 7.0*##
Female	**EVA**	Before	2.7 ± 0.3	39.0 ± 5.1
		5 weeks	3.0 ± 0.4#	43.1 ± 5.3#
		10 weeks	3.3 ± 0.3##	46.6 ± 4.5##
	PL	Before	2.3 ± 0.5	36.0 ± 5.0
		5 weeks	2.8 ± 0.5#	42.2 ± 6.2#
		10 weeks	2.9 ± 0.5##	44.3 ± 6.6##

L = litres, ml = millilitres, kg = kilograms, min. = minute

*Denotes males significantly different from females, $p<0.05$. # Denotes significantly different from before training, $p<0.05$. ## Denotes significantly different from before and after 5 weeks of training, $p<0.05$.

Table 3. The effects of elk velvet antler (EVA) and placebo (PL) supplementation on predicted bench and leg press strength before and after 5 and 10 weeks of concurrent strength and endurance training. Values are means ± standard deviations.

		Training	Predicted Bench Press Strength (kg)	Predicted Leg Press Strength (kg)
Male	**EVA**	Before	72.0 ± 15.6*	293.0 ± 43.1*
		5 weeks	78.3 ± 17.4*#	329.6 ± 62.1*#
		10 weeks	80.8 ± 15.1*##	344.8 ± 56.1*##
	PL	Before	79.0 ± 10.3*	293.3 ±5 6.2*
		5 weeks	80.6 ± 12.4*#	323.0 ± 61.1*#
		10 weeks	82.9 ± 11.2*##	330.1 ± 61.0*##
Female	**EVA**	Before	39.7 ± 6.3	145.3 ± 38.7
		5 weeks	42.0 ± 6.9#	173.2 ± 49.6#
		10 weeks	44.7 ± 8.4##	195.8 ± 56.7##
	PL	Before	37.9 ± 8.7	139.8 ± 69.0
		5 weeks	40.3 ± 8.1#	157.8 ± 53.2#
		10 weeks	44.3 ± 9.0##	175.9 ± 47.4##

kg = kilogram

*Denotes males significantly different from females, $p<0.05$. # Denotes significantly different from before training, $p<0.05$. ## Denotes significantly different from before and after 5 weeks of training, $p<0.05$.

Table 4. The effects of elk velvet antler (EVA) and placebo (PL) supplementation on 2000 metre rowing time before and after 5 and 10 weeks of concurrent strength and endurance training. Values are means ± standard deviations.

		Training	2000 m Rowing Time (seconds)
Male	**EVA**	Before	461.8 ± 37.3*∝
		5 weeks	437.7 ± 24.2*#∝
		10 weeks	427.6 ± 22.4*##∝
	PL	Before	463.3 ± 38.0*
		5 weeks	438.2 ± 25.6*#
		10 weeks	430.3 ± 22.1*##
Female	**EVA**	Before	515.6 ± 34.3∝
		5 weeks	490.0 ± 27.1#∝
		10 weeks	480.0 ± 21.5##∝
	PL	Before	561.1 ± 41.1
		5 weeks	523.4 ± 30.0#
		10 weeks	509.6 ± 29.7##

*Denotes males significantly different from females, $p<0.05$. # Denotes significantly different from before training, $p<0.05$. ## Denotes significantly different from before and after 5 weeks of training, $p<0.05$. ∝ Denotes significantly different from the PL group, $p<0.05$.

Table 5. The effects of elk velvet antler (EVA) and placebo (PL) supplementation on serum hormone concentrations at rest, 5 and 60 minutes after a 2000 m simulated rowing race as well as before and after 5 and 10 weeks of concurrent strength and endurance training in males. Values are ± standard deviations.

		Rest		5 min	Post Exercise	60 min	Post Exercise
Hormone	Training	EVA	Placebo	EVA	Placebo	EVA	Placebo
TT* ($ng \cdot ml^{-1}$)	Before	3.70 ± 1.38	4.23 ± 1.09	5.35 ± 1.25**	5.10 ± 0.77**	3.58 ± 0.92	3.79 ± 1.13
	5 weeks	3.46 ± 1.47	4.01 ± 0.85	5.25 ± 1.70**	5.77 ± 1.29**	3.72 ± 1.25	3.90 ± 0.90
	10 weeks	3.99 ± 1.65	3.86 ± 0.73	5.16 ± 1.90**	5.44 ± 1.16**	3.61 ± 1.38	3.82 ± 1.10
FT*($pg \cdot ml^{-1}$)	Before	18.34 ± 10.36	18.52 ± 4.76	24.01 ± 8.88**	24.92 ± 7.40**	15.91 ± 5.30	17.33 ± 6.06
	5 weeks	15.70 ± 8.11	17.65 ± 4.36	24.86 ± 9.53**	26.62 ± 7.98**	17.40 ± 7.50	17.42 ± 5.93
	10 weeks	17.48 ± 9.25	16.72 ± 3.21	27.61 ± 15.71**	22.85 ± 5.56**	16.61 ± 9.19	16.59 ± 4.95
GH• ($ng \cdot ml^{-1}$)	Before	1.01 ± 1.52	1.04 ± 1.26	13.35 ± 11.64**	12.30 ± 11.19**	6.98 ± 6.50#	8.43 ± 9.31#
	5 weeks	1.84 ± 1.57	0.63 ± 0.96	17.97 ± 16.53**	15.77 ± 10.82**	8.14 ± 10.96#	10.94 ± 10.57#
	10 weeks	0.45 ± 0.55	1.87 ± 2.16	17.27 ± 12.79**	17.41 ± 11.15**	11.16 ± 9.66#	10.80 ± 9.20#
C ($\mu g \cdot dl^{-1}$)	Before	14.88 ± 5.25	13.35 ± 4.96	19.69 ± 6.06■	18.49 ± 5.38■	18.85 ± 5.34■	18.61 ± 7.27■
	5 weeks	12.00 ± 4.69	12.68 ± 5.23	18.39 ± 5.69■□	20.55 ± 6.61■□	19.14 ± 5.25■	19.06 ± 7.67■
	10 weeks	13.49 ± 2.57	12.38 ± 4.18	20.17 ± 7.52■□	25.77 ± 6.68■□	20.58 ± 9.67■□□	21.36 ± 8.47■□□

EVA = elk velvet antler, Min. = minute, TT = total testosterone, FT = free testosterone, GH = growth hormone, C = cortisol. * = hormone was significantly different from females, $p<0.05$. ** = significantly different from Rest and 60 Min. Post Exercise, $p<0.05$. # = significantly different from Rest and 5 min. Post Exercise, $p<0.05$. • = males significantly different from females after 5 and 60 Min. Post Exercise but not at Rest, $p<0.05$. ■ = significantly different from Rest, $p<0.05$. □ = significantly different from before training, $p<0.05$. □□ = significantly different from before and after 5 weeks of training, $p<0.05$.

Table 6. The effects of elk velvet antler (EVA) and placebo (PL) supplementation on serum hormone concentrations at rest, 5 and 60 minutes after a 2000 m simulated rowing race as well as before and after 5 and 10 weeks of concurrent strength and endurance training in females. Values are ± standard deviations.

		Rest		5 min	Post Exercise	60 min	Post Exercise
Hormone	Training	EVA	Placebo	EVA	Placebo	EVA	Placebo
TT* ($ng \cdot ml^{-1}$)	Before	0.11 ± 0.09	0.14 ± 0.15	0.30 ± 0.27	0.21 ± 0.26	0.15 ± 0.14	0.18 ± 0.21
	5 weeks	0.07 ± 0.08	0.17 ± 0.20	0.18 ± 0.13	0.25 ± 0.24	0.12 ± 0.13	0.21 ± 0.21
	10 weeks	0.09 ± 0.08	0.10 ± 0.15	0.22 ± .013	0.20 ± 0.18	0.17 ± 0.13	0.20 ± 0.16
FT*($pg \cdot ml^{-1}$)	Before	1.50 ± 0.78	1.65 ± 0.80	1.87 ± 1.09	1.90 ± 0.97	1.69 ± 1.06	1.63 ± 0.76
	5 weeks	1.47 ± 0.90	1.41 ± 1.28	2.07 ± 0.89	2.13 ± 1.26	1.52 ± 0.95	1.74 ± 1.29
	10 weeks	1.30 ± 0.85	1.23 ± 0.60	1.85 ± 0.85	1.89 ± 0.88	1.88 ± 1.08	1.64 ± 0.64
GH• ($ng \cdot ml^{-1}$)	Before	1.52 ± 1.31	4.02 ± 3.87	24.61 ± 8.98**	15.40 ± 10.64**	6.52 ± 6.69	4.59 ± 3.63
	5 weeks	3.81 ± 2.80	3.02 ± 2.78	23.18 ± 11.50**	16.64 ± 9.08**	5.30 ± 3.39	4.62 ± 3.36
	10 weeks	2.49 ± 2.63	4.90 ± 4.52	18.21 ± 8.76**	19.74 ± 11.93**	5.15 ± 2.67	6.55 ± 4.39
C ($\mu g \cdot dl^{-1}$)	Before	17.80 ± 12.46	15.02 ± 4.44	22.37 ± 7.83 ■	21.35 ± 10.01 ■	23.66 ± 7.75 ■	21.04 ± 8.30 ■
	5 weeks	15.44 ± 6.15	14.79 ± 4.20	25.77 ± 13.60 ■□	27.21 ± 12.76 ■□	24.54 ± 6.45 ■	24.99 ± 10.10 ■
	10 weeks	15.22 ± 8.69	16.45 ± 12.84	27.08 ± 9.62 ■□	29.57 ± 23.36 ■□	29.84 ± 11.14 ■□	28.65 ± 18.20 ■□

EVA = elk velvet antler, Min. = minute, TT = total testosterone, FT = free testosterone, GH = growth hormone, C = cortisol. * = hormone was significantly different from females, $p<0.05$. ** = significantly different from Rest and 60 Min. Post Exercise, $p<0.05$. # = significantly different from Rest and 5 min. Post Exercise, $p<0.05$. • = males significantly different from females after 5 and 60 Min. Post Exercise but not at Rest, $p<0.05$. ■ = significantly different from Rest, $p<0.05$. □ = significantly different from before training, $p<0.05$. □□ = significantly different from before and after 5 weeks of training, $p<0.05$.

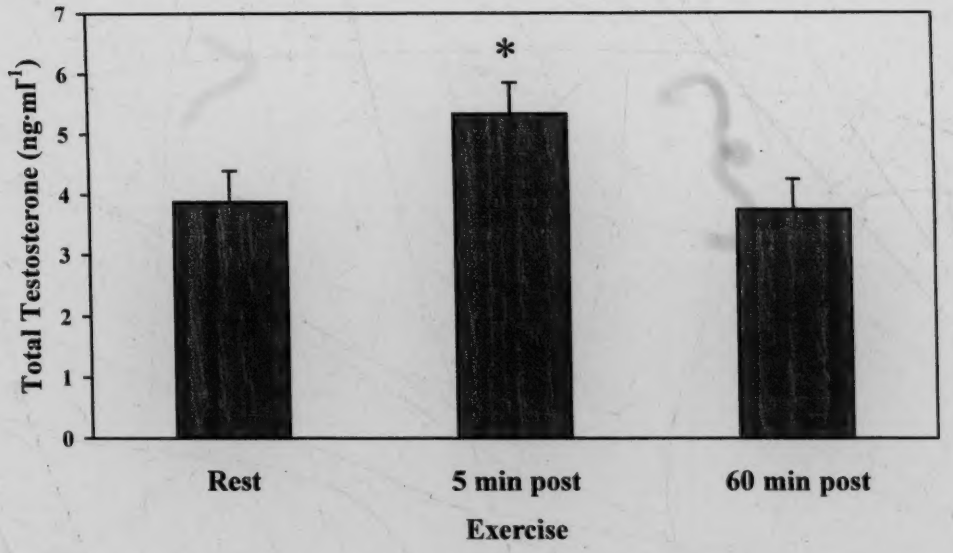

Figure 2: Serum Total Testosterone Concentrations in Males at Rest and 5 and 60 Minutes Post Exercise

*significantly different from rest and 60 minutes post exercise values, $p<0.05$

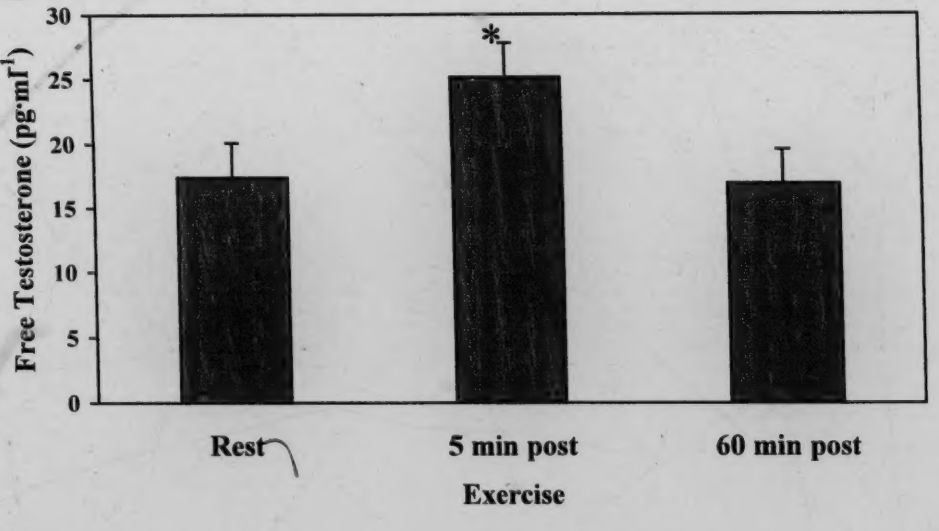

Figure 3: Serum Free Testosterone Concentrations in Males at Rest and 5 and 60 Minutes Post Exercise

*significantly different from rest and 60 minutes post exercise values, $p<0.05$

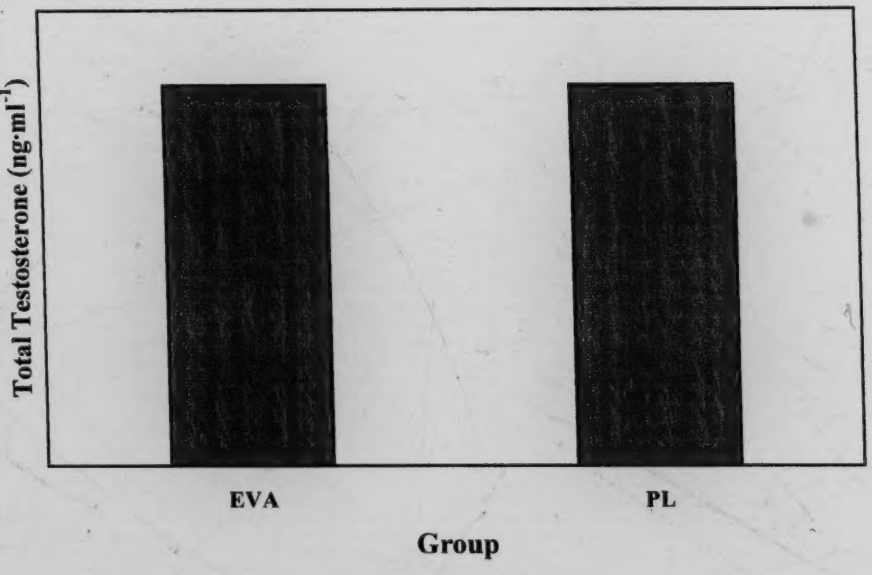

Figure 4: Main Effect for Group in Serum Total Testosterone Concentrations

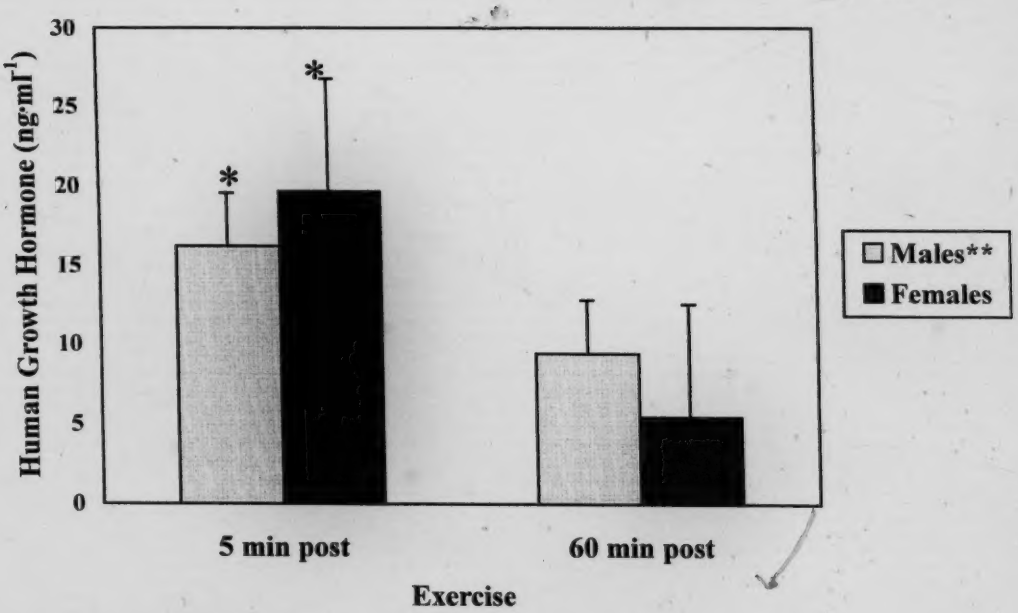

Figure 5: Serum Human Growth Hormone Concentrations in Males and Females at 5 and 60 Minutes Post Exercise

*significantly different from 60 minute post exercise values, $p<0.05$
**males significantly different from females, $p<0.05$

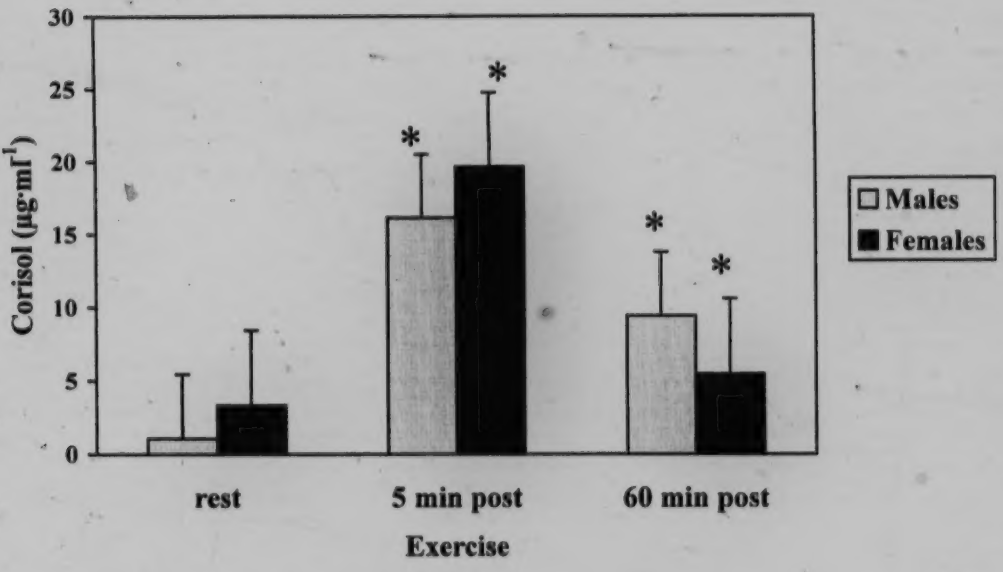

Figure 6: Main Effect for Exercise in Serum Cortisol Concentrations

*significantly different from resting values, $p<0.05$

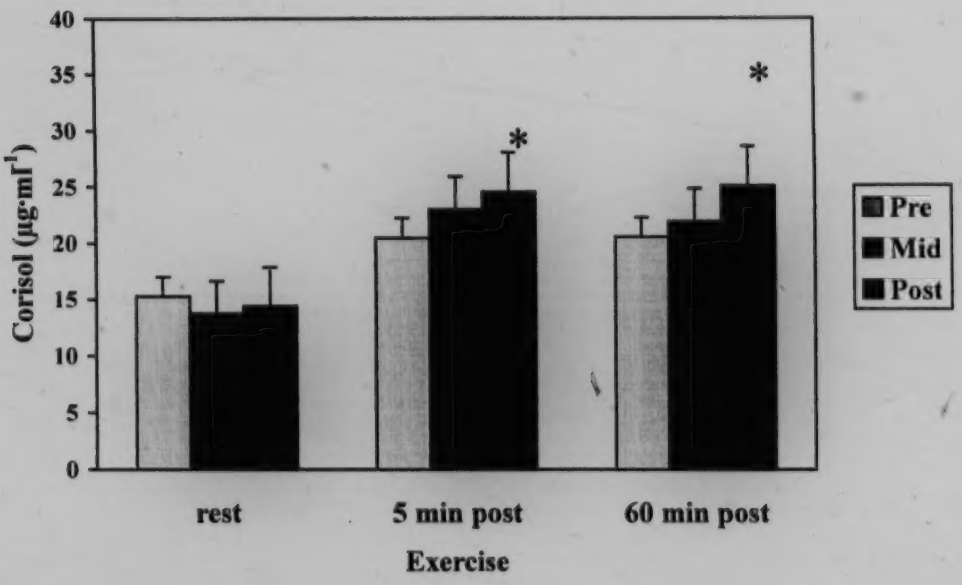

Figure 7: Interaction Effect for Exercise and Training in Serum Cortisol Concentrations

*Significantly different from pre-training 5 minutes post exercise values, $p<0.05$
**Significantly different from pre and mid-training 60 minutes post exercise values, $p<0.05$

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CHAPTER 4

GENERAL DISCUSSION AND CONCLUSION

4.1 Discussion

The findings from the present study did not support the hypothesis that subjects who supplemented with EVA while training would have enhanced performance over subjects training with a similar program, but ingesting a placebo. Furthermore, the results did not show that subjects who supplemented with EVA had an enhanced anabolic or reduced catabolic profile when compared to the placebo group, which might enable the EVA group to maintain strength development while performing concurrent strength and endurance training.

Both the EVA and PL supplementation groups (male and female) achieved similar results from the training program. All groups significantly decreased sum of skinfold scores, increased predicted leg and bench press strength and both relative and absolute VO_2 max scores after 5 and 10 weeks of concurrent strength and endurance training. Moreover, all groups significantly decreased 2000 m simulated race rowing time from baseline after 5 and 10 weeks of training. Male EVA subjects improved rowing performance from 461.8 s to 427.6 s, and male PL subjects decreased from 463.3 s to 430.3 s. Female EVA subjects improved rowing performance from 515.6 s to 480.0 s, while female PL subjects went from 561.1 s to 509.6 s. These results can be expected from the training program and are consistent with other research examining the effects of concurrent strength and endurance training (Bell et al., 1997; Bell et al., 2000; Hennessey & Watson, 1994; Hickson, 1980).

The present study demonstrated an enhanced post-test catabolic cellular environment during the recovery period following the 2000 m rowing test in all groups, as was evident by the elevated serum cortisol response immediately after exercise and which remained elevated 60 minutes post exercise, following 10 weeks of concurrent strength and endurance training. After only five weeks of training, the serum cortisol concentration was elevated at 5 minutes post exercise, but did not remain elevated at 60 minutes post exercise. This suggests that 5 weeks of concurrent strength and endurance training was not enough to create an enhanced catabolic environment during the recovery phase.

No training effect was found for serum total or free testosterone, in either group or gender, although males were found to have significantly increased serum total and free testosterone concentrations at 5 minutes post exercise when compared to baseline and 60 minutes post exercise values. This supports the work of Bell et al. (1997), who examined the effect of strength training and concurrent strength and endurance training on strength, testosterone and cortisol. Bell and colleagues measured serum hormone concentrations over the course of a 16-week training period, and observed no significant changes in total testosterone in either gender or in the strength or concurrent training groups, over the training period. The present study also demonstrated significantly increased serum testosterone concentrations following a short-term, intense bout of exercise (2000 m rowing test), which further lends support to previous research (Cumming et al., 1986; Galbo et al., 1977). No training effect was observed for either males or females in relation to serum growth hormone concentrations in the present study, findings which support the work of Pitkanen et al. (2002).

The results of the serum hormone concentrations suggest that 10 weeks of concurrent strength and endurance training does not induce an increased anabolic state, with or without EVA supplementation, which is essential in maintaining and amplifying skeletal muscle mass as a result of strength training (Bell et al., 2000; Kraemer et al., 1995). The lack of an enhanced anabolic state may be due to an increase in cortisol as a result of training (Bell et al., 1997; Bell et al., 2000; Kraemer et al., 1995) or as a result of increased metabolic clearance rates of anabolic hormones.

One limitation of the present study was that it did not compare subjects following the concurrent training program to control groups performing strength only or endurance only programs. However, we can conclude that an interference effect compromising strength development did not occur in the present study as all groups demonstrated a statistically significant linear increase in both bench and leg press strength after 5 and 10 weeks of training. The EVA group did not achieve significantly higher predicted leg or bench press scores than the PL group after training. This indicates that, even without an interference effect, the EVA supplementation did not result in greater increases in strength development over training alone.

4.2 Practical Applications

The present study did not reveal any physiological reasons to believe that 10 weeks of EVA supplementation in conjunction with combined strength and endurance training enhances rowing performance greater than training alone. The EVA group did not demonstrate significantly improved performance over the PL group in any of the physiological variables measured. Although both training groups experienced significant improvements in their 2000 m stimulated rowing times, the EVA group did not have

significantly lower 2000 m rowing performance times at either the mid (5 week) or post (10 week) testing periods when compared to the PL group. Thus, although the EVA supplementation did not appear to enhance performance, neither did the supplementation hinder performance in either group. It is possible that the anecdotal claims made by some elite athletes concerning EVA supplementation are purely psychological. From a practical perspective, short-term EVA supplementation with training does not appear to give athletes a physiological advantage to rowing performance and therefore, would not be recommended by the authors of this study as a performance aid without further research.

4.3 Future Research

Ten weeks of EVA supplementation does not appear to significantly improve rowing performance greater than training alone. Further research must be conducted before the complete effects of EVA supplementation as an ergogenic aid can be understood. The present study examined the effect of EVA on various measures of performance during concurrent strength and endurance training in rowers. Preliminary research in animals has indicated that velvet antler supplementation may increase stroke volume (Clifford et al., 1979) and plasma testosterone concentrations (Wang et al., 1988). Research in humans has reported that velvet antler supplementation may result in increased muscular strength and endurance (Sleivert et al., in press). Not all of these physiological variables were examined in the present study and must be researched in depth before it can be said if supplementing with EVA provides an advantage to performance.

In addition, research is needed to examine the dose-response relationship of EVA. As no dosage guidelines have been clinically established, we chose to follow the manufacturers recommendations for dosage at 560 mg/day. It is conceivable that this dosage was not enough to elicit a physiological response in subjects. Furthermore, research needs to be conducted into the effects of both short and long-term EVA supplementation on athletic performance.

Further, the current study investigated the effects of 10 weeks of EVA supplementation in order to correspond with the length of one term of university studies. It is possible that this was not a long enough time period for the full effects of EVA to be apparent.

Finally, research is warranted into the effects of EVA on trained and untrained populations. The population for the present study involved a physically active sample of men and women already at an elevated level of strength and rowing performance. The capacity to improve, and thus to be affected by the supplement, may be greater in a group of less experienced or more sedentary subjects.

4.4 Conclusion

The results show that EVA supplementation coupled with training did not significantly improve any of the training indices over training alone, nor did it create an enhanced anabolic or reduced catabolic cellular environment as reflected in the serum hormones measures taken in this study. Thus, there is no evidence to support an ergogenic effect for rowing performance or other training performance indices measured in this study, when ingesting EVA combined with concurrent strength and endurance training over a 10-week period in club level rowers. Additional research is needed to

further examine the potential of EVA as a supplement for athletic performance enhancement and understand its mechanisms of action before EVA should be recommended as an ergogenic aid.

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APPENDIX A

Physiological Testing

Anthropometric Measurements

Standing height was measured against a metric measuring tape that was placed vertically against a wall. The subject was instructed to stand against the wall with the heels together and heels, shoulders and buttocks touching the wall. With the subject looking straight ahead, a plane was placed on the subject's head, depressing the hair. The subject was instructed to take a deep breath, at which time the measurement was taken. A mark was made at the level of the lower border or the plane on the wall and height was recorded to the nearest 0.5 cm (PFLC Resource Manual, 1996).

Weight was measured using a Health O'Meter balance beam scale (Continental Scale Corporation, USA) and recorded in kilograms to the nearest 0.1 kg. Both height and weight were measured while the subject was not wearing footwear (PFLC Resource Manual, 1996).

Body Composition Measurements

Body composition was estimated using Harpenden skinfold callipers (Batz International, U.K.) to measure skinfold thickness at six skinfold sites and calculated using the Yuhasz method (PFLC Resource Manual, 1996). Measurements were taken at the triceps, subscapular, iliac crest, abdominal, chest and front thigh skinfold sites in men. Females were measured on the same sites, with the rear thigh being substituted for the chest skinfold site. Each skinfold site was first landmarked and then labelled with a pen. A fold was raised between the thumb and forefinger at the mark and the callipers placed perpendicular to the fold. Following a two second pause, readings were taken and

recorded to the nearest 0.2 mm. Readings were completed twice at all six sites in rotational order. If a discrepancy greater than 0.4 mm existed between the two measurements at a given site, a third measurement was taken and the mean of the two closest readings was taken as the skinfold thickness. If all three measurements were equidistant, the median measurement was used as the skinfold thickness. Percent body fat was calculated using the following formula (PFLC Resource Manual, 1996):

% Body Fat (men) = (sum of six skinfolds x 0.097) + 3.64

% Body Fat (women) = (sum of six skinfolds x 0.217) – 4.47

Landmarking for each site is as follows (PFLC Resource Manual, 1996):

Triceps: a vertical fold on the posterior surface of the upper arm halfway between the acromion of the shoulder and olecranon process of the elbow.

Subscapular: a diagonal fold running downward and laterally at about a 45 degree angle, raised directly under the inferior angle of the scapula.

Chest: a diagonal fold taken halfway between the anterior axillary line (front border of the armpit) and the nipple for men and one-third of that distance for women.

Iliac Crest: a diagonal fold running downward and anteriorly, raised directly above the crest of the ileum on the mid-axillary line.

Abdominal: a vertical fold taken about 5 cm to the left of the umbilicus.

Front Thigh: a vertical fold on the anterior thigh raised halfway between the patella and the inguinal crease with the foot on a box 20-30 cm high, the knee bent slightly and the leg relaxed.

Rear Thigh: a vertical fold on the posterior thigh at the same level as the front thigh skinfold with the leg in the same position as the front thigh skinfold.

2000 m Rowing Test

Prior to beginning the test, each subject completed a warm-up. Each test was self-paced by the subject, with the object of the test being to row 2000 m on a Concept IIC (Morrisville, VT) rowing machine in as quick a time as possible. Heart rate was monitored continuously using a Polar Pacer HR (Polar Canada, PQ) telemetry heart rate monitor. Stroke rate, 500 m split times and total time was recorded every 200 m.

Aerobic Fitness

Maximal oxygen consumption (VO_2 max) was determined by using a continuous incremental protocol to exhaustion on a Concept IIC rowing ergometer (Morrisville, VT). Female subjects began rowing at 50 W, while male subjects began at 100 W. Power output was increased by 50 W every two minutes until the subject could no longer maintain the required power output and had a respiratory exchange ratio (RER) > 1.0. At this point, the subject was instructed to go "all out", or as hard as they could, until volitional exhaustion occurred. VO_2 max was indicated by a peak and plateau (< 100 ml/min change) in VO_2 with continued exercise. Additional supportive criteria for the confirmation of VO_2 max included: RER > 1.1; heart rate at or above age predicted maximum; and, volitional exhaustion.

During the test, expired gases were collected and analyzed using a Medgraphics CPX/D Metabolic Cart (Medical Graphics, St. Paul, MN). The metabolic cart was calibrated prior to and checked after each test using known gas concentrations (Prax Air, Edmonton, AB). The subject's heart rate was continuously monitored using a Polar Pacer HR telemetry heart rate monitor system (Polar Canada, PQ). Heart rate, average strokes per minute and average power output was recorded every minute.

Strength Assessment

Strength testing for the leg and bench press exercises was conducted through the use of a multiple repetition maximum (MRM) protocol (PFLC Resource Manual, 1996). Each subject was asked to complete a set of ten repetitions at a self-selected load that was perceived to be light. A second set of ten repetitions was subsequently performed at a load that was perceived to be difficult. A third set was completed at a load selected to have the subject fail between six and ten repetitions. If the subject was able to complete ten repetitions with proper technique, a fourth set was completed to achieve failure between six and ten repetitions. A minimum of three minutes rest was given between sets. The number of repetitions the subject was able to complete until failure at the final load was used to calculate predicted 1 RM scores using Power 5.1 software (B. E. Software, Lincoln, NE).

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